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TRANSACTIONS

OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA.

THIRD SERIES.

VOLUME THE TWENTY-SIXTH.

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1904.

NOTICE.

The present volume of TRANSACTIONS contains the papers read before the College from January, 1904, to December, 1904, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

WILLIAM ZENTMAYER.

DORNAN, PRINTER.

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

1904.

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* Died May 4, 1904.

LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION.

ELECTED	
1787	JOHN REDMAN
1805	WILLIAM SHIPPEN
1809	ADAM KUHN
1818	THOMAS PARKE
1835	THOMAS C. JAMES *
1835	THOMAS T. HEWSON
1848	GEORGE B. WOOD
1879	W. S. W. RUSCHENBERGER
1883	ALFRED STILLÉ
1884	SAMUEL LEWIS †
1884	J. M. DA COSTA
1886	S. WEIR MITCHELL
1889	D. HAYES AGNEW
1892	S. WEIR MITCHELL
1895	J. M. DA COSTA
1898	JOHN ASHHURST, JR.
1900	W. W. KEEN
1902	HORATIO C. WOOD
1904	ARTHUR V. MEIGS

* Died four months after his election.
† Resigned on account of ill-health.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

DECEMBER, 1904.

* Non-resident Fellows.
† Fellows who have commuted dues.

ELECTED

*1883. Abbot, Griffith E., Ph.D., M.D., Washington, D. C.

1892. Abbott, Alex. C., M.D., Professor of Hygiene and Bacteriology, and Director of the Laboratory of Hygiene in the University of Pennsylvania.

1903. Allen, Alfred Reginald, M.D.

1873. Allis, Oscar H., M.D., Surgeon to the Presbyterian Hospital.

1896. Allyn, Herman B., M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Physician to the Philadelphia Hospital; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania.

1888. Anders, James M., M.D., LL.D., Professor of the Theory and Practice of Medicine and Clinical Medicine in the Medico-Chirurgical College; Physician to the Medico-Chirurgical and Samaritan Hospitals.

1869. Andrews, T. Hollingsworth, M.D.

1896. Angney, William M., M.D., Physician to the House of Mercy (Home for Male Consumptives); Consulting Physician to the Hospital for Diseases of the Lungs, at Chestnut Hill.

*1882. Ashbridge, Richard, M.D., West Whiteland, Pa.

1893. Ashton, Thomas G., M.D., Physician to the Philadelphia Hospital; Assistant Physician to the Jefferson Medical College Hospital; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania.

ELECTED

1857. Atlee, Walter Franklin, A.M., M.D., Corresponding Member of La Société des Sciences Médicales de Lyons; Consulting Surgeon to St. Luke's Hospital, Bethlehem; Visiting Physician to the Preston Retreat.

1852. Bache, Thomas Hewson, M.D.

1903. Bacon, John, M.D.

†1892. Baker, George Fales, B.S., M.D.

1889. Baldy, John Montgomery, M.D., Professor of Gynecology in the Philadelphia Polyclinic; Surgeon to the Gynecean Hospital and to the Gynecological Out-patient Department of the Pennsylvania Hospital; Consulting Surgeon to the Frederick Douglass Memorial Hospital.

1898. Balliet, Tilghman M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H.; Physician to the Old Man's Home.

1883. Baum, Charles, A.M., M.D., Ph.D.

1883. Beates, Henry, M.D.

1860. Benner, Henry D., M.D.

1874. Bennett, W. H., M.D., Physician-in-Charge to the Seashore Home for Invalid Children, and to the Seaside House for Invalid Women, Atlantic City; formerly Physician to the Episcopal Hospital, and Physician-in-Charge to St. Christopher's Hospital for Children.

1896. Beyea, Henry D., M.D., Associate in Gynecology and Assistant Demonstrator of Obstetrics in the University of Pennsylvania; Assistant Surgeon to the Gynecean Hospital.

†1884. Biddle, Alexander W., M.D.

1884. Biddle, Thomas, M.D.

1903. Biggs, Montgomery H., M.D., Chief Resident Physician of the Philadelphia Hospital.

1904. Birney, Herman H., M.D.

*1866. Black, J. J., M.D., New Castle, Del.

1894. Bliss, Arthur Ames, M.D., Laryngologist and Aurist to the German Hospital; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb.

*1867. Boardman, Charles H., M.D.

1894. Bochroch, Max H., M.D., Demonstrator of Neurology, and Chief Clinical Assistant in the Nervous Department of the

ELECTED

Jefferson Medical College Hospital; Neurologist to the Outpatient Department of St. Joseph's Hospital.

1896. Boger, John A., M.D., Surgeon to St. Mary's Hospital; Surgeon to the Dispensary of the Episcopal Hospital.

1891. Boyd, George M., M.D., Clinical Professor of Obstetrics in the Medico-Chirurgical College; Physician to the Philadelphia Lying-in Charity Hospital and Nurse School.

†1884. Bradford, T. Hewson, M.D.

1903. Branson, Thomas F., M.D., Attending Physician to the Bryn Mawr Hospital.

1856. Brinton, John H., M.D., LL.D., Professor of the Practice of Surgery and of Clinical Surgery in the Jefferson Medical College; Consulting Surgeon to St. Joseph's Hospital and to the Southwestern Hospital of Philadelphia.

1891. Brinton, Lewis, M.D., Visiting Physician to the Nervous Department of the Howard Hospital.

1900. Brinton, Ward, M.D., Demonstrator of Physical Diagnosis in the Jefferson Medical College; Physician to the Dispensary of St. Christopher's Hospital for Children; Assistant in the Medical Dispensary of the Jefferson Medical College Hospital.

1887. Brubaker, Albert P., M.D., Professor of Physiology in the Pennsylvania College of Dental Surgery; Adjunct Professor of Physiology and Hygiene in the Jefferson Medical College; Lecturer on Anatomy and Physiology in the Drexel Institute.

*1890. Brush, Edward N., M.D., Medical Superintendent of the Shepherd and Enoch Pratt Hospital, Towson, Md.

*1851. Bullock, William R., M.D., Wilmington, Del.

1892. Burr, Charles W., M.D., Professor of Mental Diseases in the University of Pennsylvania; Neurologist to the Philadelphia Hospital.

1886. Cadwalader, Charles E., M.D.

1895. Carpenter, John T., M.D., Lecturer on Ophthalmology in the University of Pennsylvania; Assistant Ophthalmic Surgeon to the University Hospital; Attending Ophthalmologist to the Bryn Mawr Hospital.

*1897. Carter, William S., M.D., Professor of Physiology in the University of Texas.

ELECTED

1892. CATTELL, HENRY W., A.M., M.D., Editor of Lippincott's *Medical Dictionary;* Author of *Post-mortem Pathology;* Translator of Ziegler's *Special Pathology.*

*1892. CERNA, DAVID, M.D., Ph.D., Monclova, Coah, Mexico; Corresponding Fellow of the Sociedad Española de Higiene of Madrid.

1900. CHANCE, BURTON KOLLOCK, M.D., Assistant Surgeon to the Wills Eye Hospital.

1885. CHAPIN, JOHN B., M.D., Physician-in-Chief to the Pennsylvania Hospital for the Insane.

1880. CHAPMAN, HENRY C., M.D., Professor of the Institutes of Medicine and of Medical Jurisprudence in the Jefferson Medical College.

1900. CHASE, ROBERT HOWLAND, A.M., M.D., Superintendent of the Friends' Asylum for the Insane.

1868. CHESTON, D. MURRAY, M.D.

1897. CHESTON, RADCLIFFE, M.D.

1904. CHRISTIAN, HILARY M., M.D., Clinical Professor of Genito-Urinary Diseases in the Medico-Chirurgical College; Professor of Genito-Urinary Diseases in the Philadelphia Polyclinic.

1903. CHRYSTIE, WALTER, M.D.

1899. CLARK, JOHN G., M.D., Professor of Gynecology in the University of Pennsylvania; Gynecologist-in-Chief to the University Hospital.

1897. CLAXTON, CHARLES, A.M., M.D.

1872. CLEEMANN, RICHARD A., M.D.

1896. CLEVELAND, ARTHUR H., M.D., Clinical Professor of Laryngology in the Medico-Chirurgical College; Laryngologist to the Medico-Chirurgical Hospital; Laryngologist and Aurist to the Presbyterian Hospital, and to the Pennsylvania Institution for the Deaf and Dumb.

1903. COATES, GEORGE M., M.D., Surgeon to the Dispensary of the Children's Hospital; Instructor in Otology in the Philadelphia Polyclinic; Visiting Physician to the Philadelphia Orphanage; Major and Surgeon, 2d Regiment, National Guard of Pennsylvania.

1888. COHEN, SOLOMON SOLIS, M.D., Professor of Clinical Medicine in the Jefferson Medical College; Physician to the Jefferson Medical College Hospital, to the Philadelphia Hospital, to the Jewish Hospital and to the Rush Hospital.

ELECTED

1898. Coles, Stricker, M.D., Demonstrator and Clinical Lecturer on Obstetrics in the Jefferson Medical College; Assistant Obstetrician to the Jefferson Maternity.

1901. Coley, Thomas Luther, A.B., M.D., Attending Physician to the Methodist Episcopal Hospital of Philadelphia.

1903. Cooper, J. Cardeen, M.D., Consulting Physician to the Methodist Home for the Aged.

1903. Coplin, W. M. L., M.D., Professor of Pathology in the Jefferson Medical College; Pathologist to and Director of the Laboratories of the Jefferson Medical College Hospital; Pathologist to the Philadelphia Hospital and to the Friends' Asylum for the Insane, Frankford; Bacteriologist to the Pennsylvania State Board of Health.

1904. Craig, Frank A., M.D., Assistant Instructor in Medicine in the University of Pennsylvania; Member of Staff of the Henry Phipps Institute; Examining Physician to the Free Hospital for Poor Consumptives.

1895. Cross, William A., M.D., Consulting Physician to the Jewish Hospital.

1904. Cruice, John M., M.D., Instructor in Medicine in the University of Pennsylvania; Assistant Physician to the Philadelphia Hospital; Physician to the Medical Dispensary of the University Hospital and of St. Joseph's Hospital.

1902. Currie, Charles A., M.D., Physician to the Germantown Hospital.

1884. Curtin, Roland Gideon, A.M., M.D., Ph.D., Physician to the Philadelphia and to the Presbyterian Hospitals; Consulting Physician to St. Timothy's Hospital and to the Rush Hospital for Consumptives.

1884. Da Costa, John C., M.D., Gynecologist to the Jefferson Medical College Hospital; Consulting Gynecologist to St. Agnes's Hospital; President of the Philadelphia Obstetrical Society.

1903. Da Costa, John C., Jr., M.D., Demonstrator of Clinical Medicine in the Jefferson Medical College; Chief of the Medical Clinic in the Jefferson Medical College Hospital; Assistant Physician to the Philadelphia Hospital; Hæmatologist to the German Hospital.

ELECTED

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia and to St. Joseph's Hospitals.

1887. Daland, Judson, M.D., Professor of Clinical Medicine in the Medico-Chirurgical College; Physician to the Medico-Chirurgical Hospital; Physician to the Philadelphia Hospital.

1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital.

1896. Davis, Charles N., M.D., Dermatologist to the Pennsylvania Hospital; Consulting Dermatologist to St. Agnes's Hospital; Assistant Physician in the Dispensary for Skin Diseases in the Howard Hospital.

1888. Davis, Edward P., A.M., M.D., Professor of Obstetrics in the Jefferson Medical College and in the Philadelphia Polyclinic; Visiting Obstetrician to the Jefferson and to the Polyclinic Hospitals; Obstetrician and Gynecologist to the Philadelphia Hospital; Member of the American Gynecological Society, the American Pediatric Society, and of the International Congress of Obstetrics and Gynecology.

1889. Davis, Gwilym G., M.D. (Univ. of Penna. and Goettingen), M.R.C.S. Eng., Associate Professor of Applied Anatomy in the University of Pennsylvania; Surgeon to the Episcopal, to St. Joseph's and to the Orthopædic Hospitals.

1900. Davisson, Alex. Heron, M.D., Physician to the Dispensary of the Children's Hospital.

1894. Deaver, Harry C., M.D., Surgeon to the Episcopal and to St. Mary's Hospitals, and to St. Christopher's Hospital for Children.

1887. Deaver, John B., M.D., Surgeon-in-Chief to the German Hospital; Consulting Surgeon to the Germantown Hospital.

1892. Deaver, Richard Wilmot, M.D., Surgeon to the Germantown Hospital.

1902. Dehoney, Howard, M.D.

1885. Dercum, Francis X., M.D., Professor of Nervous and Mental Diseases in the Jefferson Medical College; Neurologist to the Philadelphia Hospital; Consulting Neurologist to St. Agnes's and to the Jewish Hospitals, and to the State Asylum for the Chronic Insane of Pennsylvania.

ELECTED

1891. Dixon, Samuel G., M.D., President and Executive Curator of the Academy of Natural Sciences of Philadelphia; Member of the Council of the American Philosophical Society; Member of the Board of Trustees of the Wistar Institute of Anatomy; Member of the Board of Managers of the Philadelphia Zoölogical Society.

1896. Donnellan, P. S., M.D., L.R.C.S. and P., Ireland; Laryngologist to St. Agnes's Hospital; Medical Examiner of the Mutual Life Insurance Company of New York.

1897. Dorland, W. A. Newman, M.D., Associate in Gynecology in the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Instructor in Obstetrics in the University of Pennsylvania.

1902. Dougherty, Sherborne W., M.D., Instructor in Physical Diagnosis in the University of Pennsylvania; Physician to the Dispensary of the German Hospital; Assistant Physician to the Methodist Hospital.

1893. Downs, Norton, M.D.

1864. Downs, R. N., M.D., Consulting Physician to the Germantown Hospital.

1902. Downs, Robert N., Jr., M.D., Surgeon to the Dispensary of the Germantown Hospital.

1864. Duer, Edward L., A.M., M.D., Gynecologist to the Presbyterian Hospital; Consulting Obstetrician to the Maternity Hospital and to the Preston Retreat.

1897. Duer, S. Naudain, M.D., Physician to the Dispensary of the Presbyterian Hospital.

1871. Duhring, L. A., M.D., Professor of Dermatology in the University of Pennsylvania.

1881. Dulles, Charles Winslow, M.D., Lecturer on the History of Medicine in the University of Pennsylvania; Surgeon to the Rush Hospital.

*1871. Dunglison, Thomas R., M.D., Rosny sous Bois (Seine), France.

1860. Dunton, William R., M.D., Consulting Physician to the Germantown Hospital.

1899. Edsall, David L., M.D., Associate in Medicine in the University of Pennsylvania; Associate of the Pepper Laboratory

ELECTED

of Clinical Medicine; Physician to the Episcopal Hospital and to St. Christopher's Hospital for Children.

*1887. Edwards, William A., M.D., Los Angeles, California; Professor of Pediatrics in the Medical Department of the University of Southern California.

1904. Elmer, Walter G., M.D., Assistant Surgeon to the Presbyterian Hospital; Surgeon to the Orthopædic Dispensary of the University Hospital; Physician to the Church Home for Children.

1896. Ely, Thomas C., A.M., M.D.

1901. Erck, Theodore A., M.D., Assistant Surgeon to the Gynecean Hospital; Associate in Gynecology in the Philadelphia Polyclinic and College for Graduates in Medicine; Gynecologist to the Frederick Douglass Memorial Hospital.

1893. Eshner, Augustus A., M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic and College for Graduates in Medicine; Physician to the Philadelphia Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Physician to the Hospital for Diseases of the Lungs, at Chestnut Hill.

1868. Evans, Horace Y., A.M., M.D.

1894. Faries, Randolph, M.D., Director of Physical Education in the Protestant Episcopal Academy.

1903. Farr, Clifford B., M.D., Pathologist and Assistant Physician to the Methodist Hospital; Instructor in Diseases of the Stomach at the Philadelphia Polyclinic; Assistant Physician in the Medical Dispensary of the Hospital of the University of Pennsylvania; Visiting Physician to the House of St. Michael, etc., for Colored Cripples.

1893. Farr, William W., M.D.

1884. Fenton, Thomas H., M.D., Medical Director and Senior Ophthalmologist to the Union Mission Hospital; Ophthalmologist to St. Vincent's Home, to the Home for Aged Couples, to the Baptist Home and to the House of the Good Shepherd.

1884. Fisher, Henry M., M.D., Physician to the Out-patient Department of the Pennsylvania Hospital.

*1900. Flexner, Simon, M.D., Director of the Rockefeller Institute, New York City; Member of the Association of American Physicians; Corresponding Member of La Societa Medico-Chirurgica di Bologna.

ELECTED

1888. Flick, Lawrence F., M.D.

1862. Forbes, William S., M.D., Professor of Anatomy in the Jefferson Medical College; Clinical Surgeon to the Jefferson Medical College Hospital.

*†1885. Fox, Joseph M., M.D., Leesburg, Va.

1903. Francine, Albert P., M.D., Instructor in Medicine in the University of Pennsylvania; Member of the Staff of Phipps Institute; Medical Registrar to the Philadelphia Hospital.

1897. Frazier, Charles H., M.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University, to the Philadelphia and to the Howard Hospitals; Surgeon to the Home for Crippled Children.

†1890. Freeman, Walter J., M.D., Professor of Laryngology in the Philadelphia Polyclinic; Laryngologist to the Children's and Orthopædic Hospitals; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb.

1893. Friebis, George, M.D., Ophthalmic Surgeon to the Lutheran Home and Orphanage, Mt. Airy.

1899. Furness, William H., 3d, M.D.

1889. Fussell, M. Howard, M.D., Assistant Professor of Medicine in the University of Pennsylvania; Physician to St. Timothy's Hospital; Physician to St. Mary's Hospital.

1899. Gamble, Robert G., M.D., one of the Attending Physicians to the Bryn Mawr Hospital.

1873. Gerhard, George S., M.D., Physician-in-Chief to the Bryn Mawr Hospital; Consulting Physician to Bryn Mawr College; Consulting Physician to Villa Nova College.

1864. Getchell, F. H., M.D.

1902. Ghriskey, Albert A., M.D., Clinical Pathologist to the Episcopal Hospital.

1892. Gibb, Joseph S., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Surgeon to the Ear, Nose and Throat Department of the Episcopal Hospital.

1899. Gibbon, John H., M.D., Surgeon to the Pennsylvania Hospital; Surgeon to the Bryn Mawr Hospital; Consulting Surgeon to the Woman's Hospital; Associate Professor of Surgery in the Jefferson Medical College.

ELECTED

1897. Girvin, John H., M.D., Physician for Diseases of Women at the Presbyterian Hospital; Instructor in Obstetrics in the University of Pennsylvania.

1889. Githens, William H. H., M.D., Visiting Physician to the Sheltering Arms.

1894. Gleason, E. B., M.D., Clinical Professor of Otology in the Medico-Chirurgical College; Surgeon-in-Charge of the Nose, Throat, and Ear Department of the Northern Dispensary.

1884. Godey, Harry, M.D.

1893. Goodell, W. Constantine, M.D.

†1897. Gould, George M., A.M., M.D.

1894. Graham, Edwin E., M.D., Professor of Pediatrics in the Jefferson Medical College; Pediatrist to the Jefferson and to the Philadelphia Hospitals; Physician to the Franklin Reformatory Home.

1885. Graham, John, M.D.

1904. Grayson, Charles P., M.D., Clinical Professor of Laryngology and Rhinology in the University of Pennsylvania; Physician-in-Charge of Throat and Nose Department of the University Hospital; Oto-Laryngologist to the Philadelphia Hospital.

1883. Griffith, J. P. Crozer, M.D., Clinical Professor of the Diseases of Children in the University of Pennsylvania; Physician to St. Agnes's, to the Children's and to the Methodist Hospitals.

*1889. Guitéras, John, M.D.

1902. Gwyn, Norman B., M.B., Instructor in Medicine in the University of Pennsylvania.

*1893. Hamill, Robert H., M.D., Summit, N. J.

1894. Hamill, Samuel McC., M.D., Professor of Diseases of Children in the Philadelphia Polyclinic and College for Graduates in Medicine; Pediatrist to the Howard Hospital.

1897. Hand, Alfred, Jr., M.D., Visiting Physician to the Children's Hospital, to the Children's Hospital of the Mary J. Drexel Home of Philadelphia and to the Methodist Hospital.

1886. Hansell, Howard F., M.D., Professor of Ophthalmology in the Jefferson Medical College; Ophthalmic Surgeon to the Philadelphia Hospital and to the Jefferson Medical Col-

ELECTED

lege Hospital; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic.

1889. HARE, HOBART A., M.D., Professor of Therapeutics in the Jefferson Medical College; Physician to the Jefferson Medical College Hospital.

1865. HARLAN, GEORGE C., M.D., Consulting Surgeon to the Wills Eye Hospital; Ophthalmic Surgeon to Pennsylvania Hospital, and to the Pennsylvania Institution for the Blind; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic.

1902. HARLAND, WILLIAM G. B., M.D., Instructor in Laryngology in the University of Pennsylvania; Aurist and Laryngologist to the Dispensary of the Presbyterian Hospital; Laryngologist to the Phipps Institute for Tuberculosis; Visiting Physician to the Philadelphia Home for Incurables.

1903. HART, CHARLES D., M.A., M.D., Physician to the Out-patient Department of the Pennsylvania Hospital; Inspector of the Eastern Penitentiary.

1885. HARTE, RICHARD H., M.D., Surgeon to the Pennsylvania Hospital; Consulting Surgeon to St. Mary's and to St. Timothy's Hospitals.

1888. HARTZELL, MILTON B., M.D., Associate in Dermatology in the University of Pennsylvania; Dermatologist to the Methodist Episcopal Hospital.

1872. HAYS, I. MINIS, M.D.

1882. HEARN, W. JOSEPH, M.D., Professor of Clinical Surgery in the Jefferson Medical College; Surgeon to Philadelphia Hospital.

1901. HEISLER, JOHN C., M.D., Professor of Anatomy in the Medico-Chirurgical College.

1884. HENRY, FREDERICK P., M.D., Physician to the Philadelphia Hospital; Professor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania.

1903. HENRY, J. NORMAN, M.D., Physician to the Out-patient Department of the Pennsylvania Hospital; Assistant Physician to the Philadelphia Hospital; Clinical Instructor in Medicine in the Woman's Medical College of Pennsylvania; Physician-in-Charge of the De Lancey School.

1891. HEWSON, ADDINELL, A.M., M.D., Demonstrator of Anatomy in the Jefferson Medical College; Professor of Anatomy in

ELECTED

the Philadelphia Polyclinic and College for Graduates in Medicine; Surgeon to St. Timothy's Hospital, Roxborough; Senior Surgeon to the Dispensary of the Episcopal Hospital.

1897. HINCKLE, WILLIAM M., M.D., Lecturer on the Anatomy and Physiology of the Vocal Organs in the National School of Elocution and Oratory.

1872. HINKLE, A. G. B., M.D.

*1892. HINSDALE, GUY, A.M., M.D., Hot Springs, Virginia, Secretary of the American Climatological Association; Corresponding Member of the British Balneological and Climatological Society; ex-President of the Pennsylvania Society for the Prevention of Tuberculosis.

1888. HIRSH, A. BERN., M.D., Physician to the Home for Aged Couples.

1888. HIRST, BARTON COOKE, M.D., Professor of Obstetrics in the University of Pennsylvania; Gynecologist to the Philadelphia and to the Howard Hospitals.

1903. HIRST, JOHN COOKE, M.D., Assistant Obstetrician to the University and the Philadelphia Hospitals; Attending Physician to the Maternity Hospital and to the Gynecological Dispensary of the Howard Hospital; Consulting Obstetrician to the Southeastern Dispensary.

*1894. HOCH, WILLIAM R., M.D., Pueblo, Col.

1885. HOLLAND, JAMES W., M.D., Professor of Medical Chemistry and Toxicology in the Jefferson Medical College.

1888. HORWITZ, ORVILLE, M.D., Professor of Genito-urinary Diseases in the Jefferson Medical College; Surgeon to the Jefferson Medical College Hospital, to the Philadelphia Hospital and to the State Hospital for the Insane; Consulting Surgeon to the Jewish Hospital.

1892. HUGHES, WILLIAM E., M.D., Visiting Physician to the Philadelphia Hospital; Pathologist to the Presbyterian Hospital.

1898. HUTCHINSON, JAMES P., M.D., Surgeon to the Pennsylvania, to the Children's, to the Methodist, to St. Timothy's and to the Bryn Mawr Hospitals.

1871. INGHAM, JAMES V., M.D.

ELECTED

*1885. JACKSON, EDWARD, M.D., Denver, Col., Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic; Ophthalmologist to the County Hospital, St. Anthony's Hospital, and the Mercy Sanitarium, Denver.

1887. JAYNE, HORACE, M.D., Ph.D., Professor of Zoölogy in the University of Pennsylvania; Director of the Wistar Institute of Anatomy and Biology.

1898. JOHNSON, RUSSELL H., A.B. (Princeton), M.D., Physician to the Pennsylvania Institution for the Deaf and Dumb.

1900. JONES, CHARLES JAMES, A.M., M.D., Ophthalmic Surgeon to the House of the Good Shepherd, Germantown; Assistant Surgeon to the Wills Eye Hospital.

1900. JOPSON, JOHN H., M.D., Surgeon to the Children's Hospital, to the Bryn Mawr Hospital and to the Philadelphia Home for Incurables; Surgeon to the Dispensary of the Presbyterian Hospital.

1885. JUDD, LEONARDO DA VINCI, M.D.

1900. JUDSON, CHARLES F., A.B., M.D., Physician to the Episcopal Hospital, to St. Christopher's Hospital for Children, to the Southern Home for Destitute Children and to the Sheltering Arms.

1902. JUMP, HENRY D., M.D., Instructor in Medicine in the University of Pennsylvania; Assistant Physician to the Medical Dispensary of the University Hospital.

1886. JURIST, LOUIS, M.D.

1903. KALTEYER, FREDERICK J., M.D., Demonstrator of Clinical Medicine in the Jefferson Medical College; Chief of the Out-patient Department, Assistant Attending Physician and Hæmatologist to the Jefferson Medical College Hospital; Pathologist to the Philadelphia Lying-in-Charity.

1904. KANE, J. A. BAYARD, M.D.

†1867. KEEN, WILLIAM W., M.D., LL.D., F.R.C.S. (Hon.), Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Membre Correspondant Étranger de la Société de Chirurgie de Paris; Honorary Member of the Société Belge de Chirurgie and of the Clinical Society of London; Ehrenmitglied der Deutschen Gesellschaft für Chirurgie.

ELECTED

1897. Kelly, Aloysius O. J., M.D., Associate in Medicine in the University of Pennsylvania; Assistant Physician to the University Hospital; Clinical Professor of Pathology in the Woman's Medical College of Pennsylvania; Visiting Physician to St. Mary's and to St. Agnes's Hospitals; Pathologist to the German Hospital.

*1887. Kelly, Howard A., M.D., Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md.

1898. Kempton, Augustus F., M.D.

1902. King, William Howard, M.D.

1895. Kneass, Samuel S., M.D., Associate in the William Pepper Laboratory of Clinical Medicine in the University of Pennsylvania.

1904. Krauss, Frederick, M.D., Ophthalmologist to the Dispensary of St. Christopher's Hospital for Children.

1900. Krusen, Wilmer, M.D., Instructor in Gynecology in Jefferson Medical College; Assistant Gynecologist and Chief of the Gynecological Dispensary of St. Joseph's Hospital; Consulting Gynecologist to the Children's Hospital in Germantown.

1897. Kyle, D. Braden, A.M., M.D., Professor of Laryngology in the Jefferson Medical College; Consulting Laryngologist, Rhinologist, and Otologist to St. Agnes's Hospital and to the Philadelphia Home for Incurables; Laryngologist to the New Jersey Training School for Feeble-minded Children; Bacteriologist to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1865. La Roche, C. Percy, M.D.

*1892. Laine, Damaso T., M.D., Havana, Cuba.

1904. Landis, Henry R. M., M.D., Visiting Physician to the White Haven Sanatorium; Member of the Staff of the Henry Phipps Institute; Assistant Visiting Physician to the Philadelphia Hospital.

1904. Le Boutillier, Theodore, M.D., Physician to the Dispensary of St. Christopher's Hospital for Children; Instructor in Diseases of Children in the Philadelphia Polyclinic; Registrar to the Medical Dispensary of the Children's Hospital; Visiting Physician to the Baptist Orphanage and Haddock Memorial Home.

ELECTED

1893. Le Conte, Robert G., A.B., M.D., Surgeon to the Pennsylvania, to the Children's and to the Bryn Mawr Hospitals; Consulting Surgeon to the Germantown Hospital.

1887. Leaman, Henry, M.D.

1903. Leffmann, Henry, A.M., M.D., D.D.S., Ph.D., Professor of Chemistry and Toxicology in the Woman's Medical College of Pennsylvania; Professor of Chemistry in the Wagner Free Institute of Science; Pathological Chemist to the Jefferson Medical College Hospital; Chemist to the State Board of Health of Pennsylvania.

1892. Leidy, Joseph, M.D., Physician to the Out-patient Department of the Pennsylvania Hospital; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children.

1903. Leonard, Charles Lester, M.D.

1877. Lewis, Morris J., M.D., Physician to the Children's Hospital, to the Orthopædic Hospital and Infirmary for Nervous Diseases and to the Pennsylvania Hospital.

1904. Lindauer, Eugene, M.D., Instructor in Clinical Medicine in the Philadelphia Polyclinic.

1886. Lloyd, J. Hendrie, M.D., Neurologist to the Philadelphia Hospital; Physician to the Methodist Episcopal Hospital and to the Home for Crippled Children; Consulting Neurologist to the State Asylum for the Chronic Insane of Pennsylvania and to the Pennsylvania Training School for Feeble-minded Children.

1900. Lodge, John W., M.D., Consulting Physician to the Bryn Mawr Hospital.

1893. Longaker, Daniel, M.D., Surgeon to the Jewish Maternity Hospital.

1877. Longstreth, Morris, M.D.

1900. McCarthy, Daniel J., M.D., Instructor in Neurology in the Philadelphia Polyclinic; Associate of the William Pepper Laboratory of Clinical Medicine.

1875. McClellan, George, M.D., Consulting Surgeon to the Howard Hospital.

1903. McConnell, Guthrie, M.D., Demonstrator of Pathology in the Medico-Chirurgical College; Assistant Pathologist to the

ELECTED

Philadelphia Hospital; Pathologist to the Howard Hospital; Assistant Pathologist to the Polyclinic Hospital.

1895. McFarland, Joseph, M.D., Professor of Pathology and Bacteriology in the Medico-Chirurgical College; Pathologist to the Medico-Chirurgical and the Philadelphia Hospitals.

1900. McReynolds, Robert Phillips, M.D., one of the Resident Chiefs and one of the Chiefs of the Gynecological Dispensary of the Presbyterian Hospital.

1886. MacCoy, Alexander W., M.D., Surgeon for Diseases of the Nose and Throat in the Out-patient Department of the Pennsylvania Hospital; Member of the American Laryngological Society.

1901. MacLeod, George I., Jr., M.D., Assistant Physician and Registrar to the Bryn Mawr Hospital.

1896. Makuen, G. Hudson, M.D., Professor of Defects of Speech in the Philadelphia Polyclinic; Laryngologist to St. Mary's Hospital and to the Frederick Douglass Memorial Hospital; Visiting Consultant on Defects of Speech to the New Jersey Training School for Feeble-minded Children.

*1885. Mallet, John William, M.D., Ph.D. (Goett.), LL.D. (Princeton and Johns Hopkins), F.R.S., Professor of Chemistry in the University of Virginia.

1898. Marshall, George Morley, M.D., Laryngologist to the Philadelphia Hospital; Physician and Laryngologist to St. Joseph's Hospital.

1893. Marshall, John, M.D., Nat. Sc. D., LL.D., Professor of Chemistry and Toxicology in the University of Pennsylvania.

1889. Martin, Edward, M.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to University, Howard, St. Agnes's, Philadelphia, and Bryn Mawr Hospitals.

1885. Mays, Thomas J., M.D., Visiting Physician to the Rush Hospital.

*1868. Mears, J. Ewing, M.D.

1875. Meigs, Arthur V., M.D., Physician to the Pennsylvania Hospital; Consulting Physician to the Pennsylvania Institution for the Instruction of the Blind.

*1884. Mifflin, Houston, M.D., Columbia. Pa.

1894. Miller, D. J. Milton, M.D., Visiting Physician to the Episcopal and to the Children's Hospitals.

ELECTED

1881. Mills, Charles K., M.D., Professor of Neurology in the University of Pennsylvania; Neurologist to the Philadelphia Hospital.

1904. Mitchell, Charles F., M.D., Surgeon to the Out-patient Department of the Pennsylvania Hospital; Assistant Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases; Associate in Surgery in the Philadelphia Polyclinic.

†1888. Mitchell, John K., M.D., Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Neurologist to the Presbyterian Hospital; Attending Physician to the Pennsylvania Training School for Feeble-minded Children.

1856. Mitchell, S. Weir, M.D., M. Nat. Ac. Sci., LL.D. (Edinburgh), Corresponding Member of French Academy of Medicine; Assembly für innere Medicin, Berlin; Member of the Royal Medical Society.

1882. Montgomery, Edward E., M.D., Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Jefferson Medical College Hospital and to St. Joseph's Hospital.

1863. Morehouse, George Read, M.D., Ph.D. (Princeton), Consulting Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1886. Morris, Caspar, M.D.

1893. Morris, Elliston J., M.D., Physician to the Episcopal Hospital, to the Sheltering Arms and to the Midnight Mission.

1883. Morris, Henry, M.D., Professor of Anatomy in the Woman's Medical College of Pennsylvania; Senior Visiting Physician to St. Joseph's Hospital; Associate Member, Military Surgeons of U. S. A.

1856. Morris, J. Cheston, M.D.

1897. Morton, Samuel W., M.D.

1891. Morton, Thomas S. K., M.D.

1864. Moss, William, M.D.

1904. Moulton, Albert R., M.D., Senior Assistant Physician to the Pennsylvania Hospital for the Insane; Physician to the Out-patient Department for Nervous and Mental Diseases at the Pennsylvania Hospital; Professor of Mental Diseases in the Maine Medical School (Bowdoin College).

ELECTED

1882. Musser, John H., M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the University, the Philadelphia and the Presbyterian Hospitals.

1896. Myers, T. D., M.D., Lecturer on Ophthalmology in the U. S. Navy Medical School.

1902. Nassau, Charles F., M.D., Surgeon to St. Joseph's Hospital; Physician to Dispensary for Women of the German Hospital.

1886. Neff, Joseph S., M.D.

1887. Neilson, Thomas Rundle, M.D., Surgeon to the Episcopal Hospital and to St. Christopher's Hospital for Children; Clinical Professor of Genito-Urinary Diseases in the University of Pennsylvania.

1899. Nicholson, William R., Jr., M.D., Assistant Instructor in Obstetrics in the University of Pennsylvania; Obstetrician to Maternity Hospital; Assistant Surgeon to Gynecean Hosbital.

1889. Noble, Charles P., M.D., Surgeon-in-Chief to the Kensington Hospital for Women; Surgeon-in-Charge of the Department for Women of the Northern Dispensary and of the Union Mission Hospital; Clinical Professor of Gynecology in the Woman's Medical College of Pennsylvania; Lecturer on Gynecology in the Philadelphia Polyclinic.

1893. Noble, William H., M.D.

1898. Nolan, Edward J., M.D., Recording Secretary and Librarian of the Academy of Natural Sciences of Philadelphia.

1901. Norris, Henry, M.D., Chief of the Surgical Dispensary of the Hospital of the University of Pennsylvania; Clinical Assistant in the Clinic for Genito-Urinary Surgery in the Polyclinic Hospital.

1869. Norris Herbert, M.D.

*1865. Norris, Isaac, M.D.

1892. Norris, Richard C., M.D., Lecturer on Clinical and Operative Obstetrics in the University of Pennsylvania; Obstetrician in Charge to the Preston Retreat; Visiting Obstetrician to the Philadelphia Hospital; Gynecologist to the Methodist Hospital and Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital.

ELECTED

1884. Oliver, Charles A., A.M., M.D., Surgeon to the Wills Eye Hospital; Ophthalmic Surgeon to the Philadelphia and to the Presbyterian Hospitals; Associate Clinical Professor of Ophthalmology in the Woman's Medical College of Pennsylvania.

1884. O'Neill, J. Wilks, M.D.

*1885. Osler, William M.D., Professor of Medicine in the Johns Hopkins University and Physician to the Johns Hopkins Hospital, Baltimore, Md.

1903. Ostheimer, Maurice, M.D., Instructor in Pediatrics in the University of Pennsylvania; Physician to the Medical Dispensary of St. Christopher's Hospital for Children; Registrar in the Medical Dispensary of the Children's Hospital.

1897. Packard, Francis R., M.D., Aurist to the Out-patient Department of the Pennsylvania Hospital; Professor of Diseases of the Ear in the Philadelphia Polyclinic and College for Graduates in Medicine; Consulting Aurist to the Bryn Mawr Hospital; Editor of the *American Journal of the Medical Sciences.*

1858. Packard, John H., M.D., Late Surgeon to the Pennsylvania Hospital; Surgeon Emeritus to St. Joseph's Hospital.

1898. Page, Henry F., M.D., Assistant Physician to the German Hospital and Physician to the Medical Dispensary of the same; Instructor in Clinical Medicine in the Woman's Medical College of Pennsylvania; Physician to the Baptist Home.

1899. Parke, William E., M.D., Assistant Surgeon in the Department for Diseases of Women in the Northern Dispensary; Clinical Assistant and Surgeon to the Dispensary of the Kensington Hospital for Women.

*1903. Pearce, Richard M., M.D., Director of the Bender Laboratory, Albany, N. Y.; Professor of Pathology in the Albany Medical School; Director of the Bureau of Pathology and Bacteriology of the New York State Board of Health.

†1889. Penrose, Charles Bingham, M.D., Ph.D. (Harvard), Formerly Professor of Gynecology in the University of Pennsylvania.

1854. Penrose, R. A. F., M.D., LL.D., Professor (Emeritus) of Obstetrics and Diseases of Women and Children in the University of Pennsylvania.

ELECTED

†1902. Pepper, William, M.D., Instructor in Medicine in the University of Pennsylvania.

1884. Perkins, Francis M., M.D., Ophthalmic Surgeon to St. Agnes's Hospital.

1899. Phillips, John L., M.D.

1883. Piersol, George A., M.D., Professor of Anatomy in the University of Pennsylvania.

1872. Porter, William G., M.D., Surgeon to the Presbyterian Hospital; Consulting Physician to the Philadelphia Dispensary and to the Educational Home for Boys.

1896. Posey, William Campbell, M.D., Surgeon to the Wills Eye Hospital; Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Ophthalmic Surgeon to the Howard Hospital; Ophthalmologist to the Pennsylvania Hospital for Epileptics.

1902. Potts, Barton H., M.D., Laryngologist and Aurist to the German and to St. Mary's Hospitals; Assistant Aural Surgeon and Chief of the Ear Dispensary of the Children's Hospital.

1899. Potts, Charles S., M.D., Associate in Neurology in the University of Pennsylvania; Assistant Neurologist to the University Hospital; Neurologist to the Philadelphia Hospital; Consulting Physician to the Hospital for the Insane, Atlantic County, New Jersey.

†1899. Price, Joseph, M.D., Obstetric Physician to the Philadelphia Dispensary.

1903. Purves, George Morehouse, M.D., Registrar of the Surgical Dispensary of the Hospital of the University of Pennsylvania.

1903. Pyle, Walter L., M.D., Assistant Surgeon to the Wills Eye Hospital.

1887. Randall, B. Alexander, M.A., M.D., Clinical Professor of Diseases of the Ear in the University of Pennsylvania; Eye and Ear Surgeon to the Children's Hospital; Consulting Aurist to the Pennsylvania Institution for the Deaf and Dumb and to St. Timothy's Hospital.

1904. Ravenel, Mazyck P., M.D., Assistant Medical Director and Attending Physician to the Henry Phipps Institute.

ELECTED

1887. Reed, Charles H., M.D.

1885. Reichert, Edward T., M.D., Professor of Physiology in the University of Pennsylvania.

1897. Rhein, John H. W., M.D., Neurologist to St. Agnes's Hospipital; Physician to the Philadelphia Home for Incurables; Bacteriologist to the Pennsylvania Training School for Feeble-minded Children.

1891. Rhoads, Edward G., M.D.

1898. Riesman, David, M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic; Associate in Medicine in the University of Pennsylvania; Physician to the Philadelphia and to the Jewish Hospitals.

1895. Ring, G. Oram, M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmic and Aural Surgeon to the Samaritan Hospital.

1891. Risley, S. D., M.D., Attending Surgeon to the Wills Eye Hospital; Professor (Emeritus) of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Member of the Board of Managers of the Pennsylvania Training School for Feeble-minded Children; Alumnus Manager of the University Hospital.

†1878. Roberts, John B., M.D., Professor of Surgery in the Philadelphia Polyclinic; Surgeon to the Methodist Hospital.

1899. Roberts, Walter, M.D., Instructor in Otology in the Philadelphia Polyclinic; Clinical Assistant for Diseases of the Nose and Throat in the Out-patient Department of the Pennsylvania Hospital; Laryngologist to the Philadelphia Hospital.

1903. Rorertson, William Egbert, M.D., Assistant Visiting Physician and Chief of Dr. Daland's Clinic in the Medico-Chirurgical Hospital; Lecturer to Nurses in the Medico-Chirurgical Hospital; Pathologist to the Episcopal Hospital.

*1888. Robins, Robert P., M.D.

1902. Robinson, James Weir, M.D., Assistant Surgeon to the Presbyterian Hospital.

1903. Robinson, William Duffield, M.D.

1900. Rodman, William L., M.D., Professor of the Principles of Surgery and Clinical Surgery in the Medico-Chirurgical College; Professor of the Principles and Practice of Surgery in the Woman's Medical College of Pennsylvania.

ELECTED

1898. Ross, George G., M.D., Assistant Surgeon to the German Hospital and Surgeon to the Out-patient Department of the same; Surgeon to the Germantown Hospital.

1897. Sailer, Joseph, M.D., Associate in Medicine in the University of Pennsylvania; Associate of the Pepper Clinical Laboratory of the University of Pennsylvania; Pathologist to the Pennsylvania Training School for Feeble-minded Children.

1900. Sajous, Charles E. de M., M.D.

1899. Schamberg, Jay F., M.D., Professor of Dermatology and Infectious Eruptive Diseases in the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Physician to the Municipal Hospital for Infectious Diseases.

1887. de Schweinitz, George E., M.D., Professor of Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the Philadelphia Hospital; Consulting Ophthalmic Surgeon to the Philadelphia Polyclinic; Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1895. Scott, J. Alison, M.D., Adjunct Professor of Medicine in the University of Pennsylvania; Visiting Physician to the Pennsylvania Hospital; Physician to the Church Home for Children.

1892. Seiss, Ralph W., M.D., Professor of Otology in the Philadelphia Polyclinic; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb.

1884. Shaffner, Charles, A.M., M.D.

1897. Sharpless, William T., M.D., Physician to the Chester County Hospital, West Chester, Pa.

1876. Shippen, Edward, A.M. (Princeton), M.D., Medical Director U. S. Navy (retired).

1891. Shober, John B., A.M., M.D., Associate Gynecologist to the Gynecean Hospital; Gynecologist to the Howard Hospital; Obstetrician to the Philadelphia Hospital.

1890. Shoemaker, George Erety, A.M., M.D., Gynecologist to the Presbyterian Hospital and to the Pennsylvania Epileptic Hospital and Colony Farm.

ELECTED

†1893. SHOEMAKER, HARVEY, M.D., Visiting Physician to the Sheltering Arms and to the Southern Home for Destitute Children; Assistant Phy ician to the German Hospital; Physician to the Out-patient Department of the German and to the Pennsylvania Hospitals.

†1896. SHOEMAKER, WILLIAM T., M.D., Ophthalmic Surgeon to the Germantown Hospital; Assistant Ophthalmologist and a Chief of Clinic to the German Hospital; Dispensary Ophthalmic Surgeon to the Presbyterian Hospital; Oculist to Pennsylvania Institution for the Deaf and Dumb.

1900. SHUMWAY, EDWARD ADAMS, B.S., M.D., Instructor in Ophthalmology in the University of Pennsylvania; Assistant Ophthalmic Surgeon to the University and to the Philadelphia Hospitals; Ophthalmologist to the Henry Phipps Institute for the Study, Treatment and Prevention of Tuberculosis, to the Presbyterian Home for Widows and Single Women, and to the Magdalene Home.

1880. SIMES, J. HENRY C., M.D., Emeritus Professor of Genito-Urinary and Venereal Diseases in the Philadelphia Polyclinic.

1903. SINCLAIR, JOHN FALCONER, M.D., Physician to the Medical Dispensary of the Presbyterian Hospital; Physician to the Philadelphia Orphan Asylum and to the Presbyterian Orphanage; Physician to the Home of the Merciful Saviour for Crippled Children.

1872. SINKLER, WHARTON, M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Neurologist to the State Asulum for the Chronic Insane of Pennsylvania.

1902. SITER, E. HOLLINGSWORTH, M.D., Instructor in Genito-urinary Diseases in the University of Pennsylvania; Chief Surgeon to the Genito-urinary Dispensary of the University Hospital; Surgeon to the Dispensaries of the Children's and St. Agnes's Hospitals.

1904. SKILLERN, PENN GASKILL, M.D.

1895. SLOCUM, HARRIS A., M.D., Professor of Gynecology in the Philadelphia Polyclinic; Gynecologist to St. Clement's Hospital for Epileptics.

1904. SMITH, ALLEN J., M.D., Professor of Pathology in the University of Pennsylvania; Pathologist to the University Hospital, to the Philadelphia General Hospital and to the State Hospital for the Insane at Norristown, Pa.

ELECTED

1895. Spellissy, Joseph M., A.M., M.D., Surgeon to the Outpatient Department of the Pennsylvania Hospital; Visiting Surgeon to St. Joseph's and to the Methodist Episcopal Hospitals; Assistant Surgeon to the Orthopædic Department of the University Hospital.

1897. Spiller, William G., M.D., Associate Professor of Neurology and Professor of Neuropathology in the University of Pennsylvania; Clinical Professor of Nervous Diseases in the Woman's Medical College of Pennsylvania; Professor of Nervous Diseases in the Philadelphia Polyclinic and College for Graduates in Medicine.

1894. Stahl, B. Franklin, Ph.G., B.S., M.D., Instructor in Physical Diagnosis and Lecturer on Dietetics of the Sick in the University of Pennsylvania; Assistant Physician to the Philadelphia Hospital; Medical Director of St. Agnes's Hospital.

1904. Stanton, William B., M.D., Physician to the Department of Tuberculosis, Philadelphia Hospital; Physician to the Henry Phipps Institute; Visiting Physician to the Free Hospital for Poor Consumptives, White Haven, Pa.

1875. Starr, Louis, M.D.

1898. Steele, J. Dutton, M.D., Associate in Medicine in the University of Pennsylvania; Physician to the Medical Dispensary of the University Hospital; Bacteriologist to the Presbyterian Hospital.

1892. Steinbach, Lewis W., M.D., Professor of Clinical and Operative Surgery in the Philadelphia Polyclinic; Visiting Surgeon to the Philadelphia and to the Jewish Hospitals.

1884. Stelwagon, Henry W., M.D., Ph.D., Professor of Dermatology in the Jefferson Medical College; Dermatologist to the Philadelphia and to the Howard Hospitals; Associate Member of the Society of Dermatology and Syphilology of France and of Italy.

1895. Stengel, Alfred, M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the University, to the Pennsylvania and to the Philadelphia Hospitals.

1901. Stevens, Arthur A., M.D., Professor of Pathology in the Woman's Medical College of Pennsylvania; Lecturer on Physical Diagnosis in the University of Pennsylvania; Physician to the Episcopal and St. Agnes's Hospitals.

ELECTED

1888. Stewart, David D., M.D., Consulting Physician to the Kensington Hospital for Women.

1902. Stewart, Francis T., M.D., Professor of Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Surgeon to the Germantown Hospital; Assistant Surgeon to the Jefferson Medical College Hospital; Surgeon to the Out-patient Department of the Pennsylvania Hospital.

1898. Stout, George C., M.D., Professor of Otology in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist and Aurist to the Presbyterian Hospital, the Children's Aid Society, and to the William Penn Charter School.

1884. Stryker, Samuel S., M.D., Physician to the Presbyterian Hospital.

1900. Swan, John M., M.D., Demonstrator of Osteology in the University of Pennsylvania; Pathologist to St. Mary's Hospital.

1898. Sweet, William M., M.D., Professor of Diseases of the Eye in the Philadelphia Polyclinic; Demonstrator of Ophthalmology in the Jefferson Medical College; Assistant Ophthalmic Surgeon to the Jefferson Medical College Hospital and to the Philadelphia Hospital; Ophthalmologist to the Phœnixville Hospital.

1900. Talley, James Ely, A.B., M.D., Physician to the Out-patient Department of the Presbyterian Hospital; Assistant and Consulting Physician to the Country Branch of the Children's Hospital.

1901. Taylor, J. Gurney, M.D., Physician to the Pennsylvania Institution for the Instruction of the Blind.

1886. Taylor, John Madison, A.M. (Princeton), M.D., Editor *Monthly Cyclopædia of Practical Medicine;* Pediatric Physician to the Philadelphia Hospital; Assistant Physician to the Children's Hospital; Consulting Physician to the Elwyn, Pa., and to the Vineland, N. J., Training Schools for Feeble-minded Children.

1887. Taylor, William J., M.D., Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to St. Agnes's Hospital; Consulting Surgeon to the West Philadelphia Hospital for Women.

ELECTED

1886. Taylor, William L., M.D.

1867. Thomas, Charles Hermon, M.D.

1897. Thomson, A. G., M.D., Ophthalmic Surgeon to the Children's Hospital; Assistant Surgeon to the Wills Eye Hospital; Assistant Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases.

†1869. Thomson, William, M.D., Emeritus Professor of Ophthalmology in the Jefferson Medical College; Surgeon to Wills Eye Hospital.

1896. Thorington, James, A.M., M.D., Professor of Diseases of the Eye in the Philadelphia Polyclinic; Member of the American Ophthalmological Society; Ophthalmologist to the Vineland and Elwyn Training Schools for Feeble-minded Children.

1898. Thornton, Edward Q., M.D., Assistant Professor of Materia Medica in the Jefferson Medical College.

1896. Toulmin, Harry, M.D., Assistant Medical Director of the Penn Mutual Life Insurance Company.

1901. Tucker, Henry, M.D.

†1894. Tunis, Joseph Price, M.D., Medical Examiner to the Equitable Life Assurance Society of the United States.

1901. Turner, John B., M.D., Clinical Assistant at the Wills Eye Hospital.

1866. Tyson, James, A.M., M.D., Professor of Medicine in the University of Pennsylvania and Physician to the Hospital of the University of Pennsylvania; Physician to the Pennsylvania Hospital.

1897. Tyson, T. Mellor, M.D., Physician to the Philadelphia Hospital; Assistant Physician to the Hospital of the University of Pennsylvania; Physician to the Rush Hospital, to the Philadelphia Lying-in Charity Hospital and to the Children's Aid Society of Philadelphia.

1904. Uhle, Alexander A., M.D., Assistant Surgeon to the Dispensary of the German Hospital; Assistant Surgeon to the Dispensary for Genito-urinary Diseases, University Hospital.

1873. Van Harlingen, Arthur, Ph.B. (Yale), M.D., Emeritus Professor of Diseases of the Skin in the Philadelphia Polyclinic; Dermatologist to the Children's Hospital.

ELECTED

1903. Van Pelt, William Turner, M.D., Ophthalmic Surgeon to the Episcopal Hospital.

1893. Vansant, Eugene Larue, M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Visiting Physician to the Throat, Nose and Ear Department of the Howard Hospital.

1897. Veasey, Clarence A., M.D., Adjunct Professor of Diseases of the Eye in the Philadelphia Polyclinic and College for Graduates in Medicine; Demonstrator of Ophthalmology in the Jefferson Medical College; Chief Clinical Assistant to the Ophthalmological Department of the Jefferson Medical College Hospital; Consulting Ophthalmologist to the Philadelphia Lying-in Charity Hospital.

†1883. Vinton, Charles Harrod, M.D.

1903. Wadsworth, William Scott, M.D.

1885. Walker, James B., M.D., Ph.D., Consulting Physician to the West Philadelphia Hospital for Women and Children.

1904. Walsh Joseph, A.M., M.D., Physician to the Henry Phipps Institute; Visiting Physician to the White Haven Sanatorium; Physician to the Department of Tuberculosis at the Philadelphia Hospital.

1893. Warren, Joseph W., M.D., Associate Professor of Physiology in Bryn Mawr College.

1895. Watson, Arthur W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Laryngologist to the Howard Hospital and to the Hospital for Diseases of the Lungs, Chestnut Hill.

1886. Watson, Edward W., M.D.

1903. Weber, Charles H., M.D., Physician to the Episcopal Hospital; Physician to the Dispensary of the Children's Hospital.

1883. Welch, William M., M.D., Physician-in-Charge of the Municipal Hospital for Contagious Diseases; Consulting Physician to the Northern Dispensary and to the Northern Home for Friendless Children.

1897. Wells, William H., M.D., Adjunct Professor of Obstetrics and Diseases of Infancy in Philadelphia Polyclinic; Demonstrator of Clinical Obstetrics in the Jefferson Medical College.

ELECTED

1893. WESTCOTT, THOMPSON S., M.D., Associate in Diseases of Children in the University of Pennsylvania; Visiting Physician to the Methodist Episcopal Hospital; Assistant Physician to the Children's Hospital.

1884. WHARTON, HENRY R., M.D., Clinical Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Presbyterian and to the Children's Hospitals; Consulting Surgeon to the Bryn Mawr Hospital, to St. Christopher's Hospital for Children and to the Pennsylvania Institution for the Deaf and Dumb.

1901. WHITE, COURTLAND Y., M.D., Assistant Director of the William Pepper Laboratory of Clinical Medicine; Lecturer on and Demonstrator of Gross Morbid Anatomy in the Veterinary Department of the University of Pennsylvania; Pathologist to the State Live Stock Sanitary Board, to the Children's, to the Jewish and to St. Joseph's Hospitals.

1878. WHITE, J. WILLIAM, M.D., Ph.D., John Rhea Barton Professor of Surgery in the University of Pennsylvania; Surgeon to the University Hospital; Fellow of the American Surgical Association and of the American Association of Genito-urinary Surgeons.

†1898. WHITING, ALBERT D., M.D., Surgeon to the Germantown Hospital; Assistant Surgeon to the German Hospital; Surgeon to the Southern Home for Destitute Children; Surgeon to the Out-patient Department of the German Hospital.

†1880. WILLARD, DE FOREST, A.M., M.D., Ph.D., Professor of Orthopedic Surgery in the University of Pennsylvania; Surgeon to the Presbyterian Hospital; ex-President of the American Surgical Association; Consulting Surgeon to the Germantown, the Atlantic City, and Phœnixville Hospitals.

*1878. WILLIAMSON, JESSE, M.D., Wilmington, Delaware, one of the Surgeons to the Delaware Hospital.

1902. WILLSON, ROBERT N., JR., M.D., Instructor in Physical Diagnosis, and Students' Physician in the University of Pennsylvania.

1881. WILSON, H. AUGUSTUS, M.D., Professor of Orthopedic Surgery in the Jefferson Medical College; Emeritus Professor of Orthopedic Surgery in the Philadelphia Polyclinic; Consulting Orthopedic Surgeon to the Philadelphia Lying-in Charity Hospital and to the Kensington Hospital for Women.

ELECTED

1874. Wilson, James C., A.M. (Princeton), M.D., Professor of the Practice of Medicine and of Clinical Medicine in the Jefferson Medical College, and Physician to the Hospital of the same (Faculty Staff); Physician-in-Chief to the German Hospital; Attending Physician to the Pennsylvania Hospital.

1902. Wilson, Samuel M., M.D.

1897. Wilson, W. Reynolds, M.D., Visiting Physician to the Philadelphia Lying-in Charity Hospital.

1904. Wister, James W., M.D., Physician to the Out-patient Department of the Germantown Hospital.

*1901. Witmer, A. Ferree, M.D., New York.

1893. Wood, Alfred C., M.D., Assistant Professor of Surgery in the University of Pennsylvania; Assistant Surgeon to the University Hospital; Surgeon to the Philadelphia and to St. Timothy's Hospitals.

1900. Wood, George B., M.D., Instructor in Laryngology in the University of Pennsylvania; Assistant in the Clinic for Diseases of the Nose and Throat at the Polyclinic Hospital.

1865. Wood, Hortaio C., M.D., LL.D. (Yale and Lafayette); Professor of Materia Medica and Therapeutics in the University of Pennsylvania; Associate Fellow in Medicine and Surgery of the American Academy of Arts and Sciences; Member of the National Academy of Science.

1903. Wood, Horatio C., Jr., M.D., Demonstrator of Pharmacodynamics in the University of Pennsylvania; Assistant Physician to the Philadelphia Hospital; Physician to the Henry Phipps Institute for the Study and Treatment of Consumption.

1880. Woodbury, Frank, M.D., Associate in Laryngology in the Philadelphia Polyclinic.

1866. Woods, D. F., M.D., Physician to the Presbyterian Hospital.

1901. Woods, Richard F., M.D., Assistant Surgeon to the Gynecean Hospital.

1888. Woodward, Charles E., M.D., Secretary of the West Chester Board of Health; U. S. Examining Surgeon; Member of the Medical Staff of the Chester County Hospital.

†1897. Woodward, George, M.D.

1903. Worden, Charles B., M.D., Associate in Diseases of the Stomach and Intestines in the Philadelphia Polyclinic; Phy-

ELECTED

sician to the Dispensary of the Presbyterian Hospital; Anæsthetizer to the Orthopedic Department of the University Hospital; Physician to the Presbyterian Orphanage.

1860. WURTS, CHARLES STEWART, M.D.

1889. YOUNG, JAMES K., M.D., Professor of Orthopedic Surgery in the Philadelphia Polyclinic; Clinical Professor of Orthopedic Surgery in the Woman's Medical College of Pennsylvania; Instructor in Orthopedic Surgery in the University of Pennsylvania and Assistant Orthopedic Surgeon to the University Hospital.

1894. ZENTMAYER, WILLIAM, M.D., Attending Surgeon to the Wills Eye Hospital; Ophthalmic Surgeon to St. Mary's Hospital; Ophthalmologist to the House of Refuge.

1899. ZIEGLER, S. LEWIS, M.D., Attending Surgeon to the Wills Eye Hospital; Ophthalmic Surgeon to St. Joseph's Hospital; Membre Société Française d'Ophtalmologie.

1887. ZIEGLER, WALTER M. L., A.M., M.D.

1895. ZIMMERMAN, MASON W., M.D., Ophthalmic Surgeon to the Germantown Hospital.

ASSOCIATE FELLOWS.

[Limited to Fifty, of whom Twenty may be Foreigners.]

American.

ELECTED

1876. Billings, John S., M.D., U. S. A. (retired), New York.
1886. Bowditch, Henry P., M.D., Boston, Massachusetts.
1877. Chaille, Stanford E., M.D., New Orleans, Louisiana.
1886. Cheever, David W., M.D., Boston, Massachusetts.
1896. Conner, Phineas Sanborn, M.D., Cincinnati, Ohio.
1893. Councilman, William T., M.D., Boston, Massachusetts.
1876. Davis, N. S., M.D., Chicago, Illinois.
1892. Emmet, Thomas Addis, M.D., New York.
1892. Fitz, Reginald H., M.D., Boston, Massachusetts.
1895. Fletcher, Robert, M.D., Washington, D. C.
1903. Gorgas, William C., M.D., U. S. A., Washington, D. C.
1891. Jacobi, A., M.D., New York.
1895. McBurney, Charles, M.D., New York.
1886. Reeve, John C., M.D., LL.D., Dayton, Ohio.
1886. Senn, Nicholas, M.D., Chicago, Illinois.
1896. Sternberg, George M., M.D., U. S. A., Washington, D. C.
1896. Tiffany, L. McLane, M.D., Baltimore, Maryland.
1894. Warren, J. Collins, M.D., Boston, Massachusetts.
1894. Weir, Robert F., M.D., New York.
1892. Welch, William H., M.D., Baltimore, Maryland.

Foreign.

1890. Baccelli, Guido, Rome, Italy.
1877. Barnes, Robert, M.D., London, England.
1894. Brunton, Sir T. Lauder, M.D., London, England.
1883. Fayrer, Sir Joseph, M.D., LL.D., F.R.S., London, England.

ELECTED

1903. Finlay, Charles J., M.D., Havana, Cuba.
1899. Fraser, Thomas R., M.D., LL.D., F.R.C.P., F.R.S., Edinburgh, Scotland.
1883. Heath, Christopher, F.R.C.S., London, England.
1903. Horsley, Sir Victor Alexander Haden, F.R.S., London, England.
1896. Jaccoud, Prof. S., M.D., Paris, France.
1874. Jackson, J. Hughlings, M.D., London, England.
1893. von Jaksch, Rudolf, M.D., Prague, Bohemia.
1903. Kocher, Prof. Theodor, M.D., Berne, Switzerland.
1896. Leyden, Ernst, M.D., Berlin, Germany.
1877. Lord Lister, M.D., LL.D., F.R.S., London, England.
1903. von Mikulicz-Radecki, Prof. J., M.D., LL.D., Breslau, Germany.
1873. Ogle, John W., M.D., London, England.
1896. Pye-Smith, P. H., M.D., London, England.
1898. Roddick, Thomas G., M.D., Montreal, Canada.
1869. Valcourt, Th. de, M.D., Cannes, France.
1904. Waldeyer, Prof. Wilhelm, M.D., Berlin, Germany.

CORRESPONDING MEMBERS.

1880. Carrow, Flemming, M.D., United States.
1880. Chiara, Domenico, M.D., Florence, Italy.
1886. Dey, Kanny Lall, M.D., Calcutta, India.
1885. Rendu, Jean, M.D., Lyons, France.

NECROLOGICAL LIST.

FELLOWS.

WASHINGTON H. BAKER,	April 1, 1904.
WILLIAM BARTON HOPKINS,	May 4, 1904.
ROBERTS BARTHOLOW,	May 20, 1904.
THOMAS M. DRYSDALE,	May 26, 1904.
F. SAVARY PEARCE,	May 27, 1904.
GEORGE M. STILES,	June 9, 1904.
AUGUSTE F. MÜLLER,	October 20, 1904.

ASSOCIATE FELLOW.

T. GAILLARD THOMAS,	February 28, 1903.

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MEMOIR OF ROBERTS BARTHOLOW, M.D.[1]

By JAMES W. HOLLAND, M.D.

It is a delicate task to prepare an acceptable memoir of a reserved man who had no intimates outside his own family. Only a close friend or relative having free access to the domestic chronicles could produce anything sufficiently sympathetic and discriminating. Under the circumstances, dependence must rest upon a few well-established facts and dates to place the subject in his environment, and let that serve as a framework for an appreciation of him in his professional capacity.

Dr. Roberts Bartholow was born at New Windsor, Maryland, on November 28, 1831. On his father's side, he came of Alsatian extraction. His mother was of English stock. The student of heredity will be interested to learn of the family tradition that the Alsatian ancestor was a Huguenot. From this class of immigrants have originated some of our foremost men. The firmness with which they held to their convictions, and their other sterling characteristics, were in no whit lessened by the hardships of the wilderness, though one could scarcely expect a survival of the social amenity which was their birthright from the polite nation. It is possible that Dr. Bartholow may have derived from this legendary source his independence, his tenacity of opinion, and his nonconformity, which were marked traits of his character, but in him there was little sign of the gay and pleasure-loving temperament of the Gaul.

He was reared in a secluded household, the loving influences of which he still cherished at the time he dedicated his treatise on *Materia Medica and Therapeutics* "To the memory of father, mother, and brothers by the survivor."

[1] Read December 7, 1904.

His parents were sufficiently well-to-do to give him a collegiate and professional education.

In 1848 he graduated in the classical course at Calvert College, now called "New Windsor College," Maryland, which was at that time under the direction of a religious brotherhood cultivating the things of the spirit "in the still air of delightful studies." In due course of time, in 1854, he received the degree of Master of Arts from the same institution.

In later life he was wont to give evidence of the sound basis of his education by apt quotations from the ancient writers. Besides this classical culture, he acquired a reading knowledge of the French language, and sufficient familiarity with the German to be able to translate medical authors. Here was laid the foundation for his accurate and refined use of our mother tongue.

Many years afterward he profited from the fact that he had acted as Assistant Instructor of Chemistry for a short while.

Immediately after taking his Bachelor's degree he began the study of medicine, and at the end of the usual curriculum received his diploma of Doctor from the Medical Department of the University of Maryland. Among his teachers were Nathan R. Smith and Samuel Chew. In the years 1855 and 1856 he was attending post-graduate courses in Baltimore. The adventurous spirit in his blood prompted him to take the competitive examination for the position of assistant surgeon in the regular army, which he passed with the highest rank. A new population was crowding into the fertile region of the West, needing protection from the fanaticism of the Mormons and from the frequent Indian outbreaks. Congress, in 1855, authorized an increase of the army, and Dr. Bartholow became a member of the medical staff under this new levy. As the culmination of many scenes of disorder, the Mormons of Utah, at the point of the "bowie" knife, had compelled the United States district judge to adjourn court *sine die*, and soon afterward he, with all other Federal officers, were forced to flee.

The Utah expedition of 1857 was sent out to protect the government officials and supersede Brigham Young, the Mormon governor. This military force was led by Colonel Albert Sidney Johnston, and on its medical staff went Roberts Bartholow, at the age of twenty-

five. In July he made the overland journey by way of the arid plains, through prairies covered with herds of countless buffalo, across the Rocky Mountains in their lonely grandeur, to meet at last the hostile and treacherous saints of Utah. Here he remained for a year, after which time he was stationed at Camp Floyd, and later at Fort Bridges, New Mexico. During the summer of 1859 he had sick leave to return home for several months. In September, 1859, he was on duty among the northwestern Indians at Fort Ridgely, Minnesota, where he remained about nine months, his next post, in 1860 and 1861, being at Fort Union, New Mexico, the region of the untamed Comanches and Apaches.

While no personal memoranda of this western life are procurable, of one thing we are assured—that the young surgeon had much experience with western fevers, with the bites of venomous reptiles, and in treating gunshot wounds, with plenty of time for thorough study of his cases. At a plastic age, he took a lasting impression from his military duties and surroundings. His naturally positive character accentuated the military manner made necessary in compelling the reckless men of the camp to obey the doctor's orders and lead clean and healthy lives.

Although the marauding red men made plentiful work for the soldiers by their numerous forays, it must have been a tiresome life, with few rational enjoyments except those derived from professional study.

In the four years of his sojourn amid scenes of territorial turmoil momentous events were happening in the East which were destined to end this formative phase of his career, and throw him into a theatre of action more worthy of his talents. In April, 1861, the Civil War was opened by the attack on Fort Sumter. Maryland, as a border slave State, at first refused to furnish troops at the President's call, but eventually was forced to fall in with the non-slaveholding States of the North.

At this time, Dr. Bartholow was stationed at Fort Union, New Mexico. Most of the officers were, like him, Southerners by birth, and all but two of them reluctantly followed their native States as they seceded in quick succession. Dr. Bartholow and one other were the only officers at that station that did not renounce their

allegiance, but in September, 1861, loyally came to Baltimore under the orders of the general government. Here he was on duty in various military hospitals for one year, after which he was sent to Fort Schuyler, New York. While in charge of the McDougall General Hospital he prepared for the use of the army *A Manual of Instruction for Enlisting and Discharging Soldiers*, which was the official standard for many years. From his own experience he wrote a book on *Qualifications for the Medical Service.* In the fall of 1863 his service was in Washington, D. C., at the Lincoln General Hospital. At this time he contributed a series of papers for the instruction of the United States Sanitary Commission, and served on a Board appointed to report upon proposed modification on "registers and returns of sick and wounded," and upon "a suitable diet to be prescribed for the general hospitals of the army." He was next on duty in the Department of the Cumberland, and at the time of his resignation had charge of the general hospital at Nashville, Tennessee.

Since the war began he had married, and his family was growing up with little chance for a settled home or a systematic education for his children. To secure these he left the army in 1864, and established himself in civil practice in Cincinnati, Ohio. He had been for seven years an army surgeon, and now, at the age of thirty-three, took up the bread-winner's duty, doubly difficult to an officer for whom the government had so long provided.

Fortunately, at this time the chair of Medical Chemistry was vacant in the Medical College of Ohio, and he was invited to take it. Having been an assistant instructor in chemistry while at college, the work was familiar and, as he performed it, not uncongenial. He was in the habit of asserting that in these positive studies he had laid the foundation of his professional education. It was not until this period of struggle for a foothold that he entered into full possession of his unusual gifts of mind.

His predecessors having been professional chemists, it is not surprising that the appointment of a practising physician was viewed with disfavor by the conservative element. But the newcomer had enthusiasm and initiative, was reckless of precedent, and won out notwithstanding the traditions he violated. The skill with which

Dr. Bartholow adjusted himself to his new duties soon justified the innovation. He kept steadily in view the applications of chemistry to medicine.

Having as yet but little private practice, he had time for investigations in sanitary and pathological chemistry. The sessions of the Academy of Medicine had moved with the even tenor of a church "experience meeting" until he disturbed it with reports on the analysis of drinking water, on sewage, ventilation, ozone, cholera excreta, etc. Here was a method of treating medical problems more convincing, as well as more arduous, than the customary one of comparing cases which brought to notice the large experience of the speakers, but left each with his opinions unchanged.

He was in charge of the Cholera Hospital during the epidemic of 1866, and later was connected with the staffs of several hospitals. He proved successful not only as a teacher, but gained daily in reputation as a practitioner of medicine. His brilliant talents had acquired greater surety without loss of vividness. By dint of practice, his literary style had grown in precision, and ultimately became admirably suited to the clearness of his thought.

His pen was never idle. Having founded the medical journal called *The Clinic*, by his trenchant criticisms he provoked controversies some of which were bitter, and in all of which he proved his mettle. His readers found a serious fascination in his editorials, in which, with an audacious hand, he held up to gaze the mistakes and follies of his contemporaries. It must be admitted that he enjoyed this form of battle, for after each attack he gained in strength. Needless to say, he raised up a host of enemies, but in spite of them, by flank movement and frontal charge, he stormed his way to victory.

He was transferred to the chair of Materia Medica in 1867, and, with characteristic zeal, soon made himself famous as an exponent of the methods of the exact sciences applied to the action of medicines. For the first time in that institution the lectures were illustrated with experiments on animals demonstrating the physiological effects of drugs.

In the maturity of his powers he was amazingly busy, being pathologist to one hospital, clinical lecturer to another, professor,

editor, and active practitioner. While engrossed with these multifarious pursuits, he yet found time to write a book on *Disinfection*, one on *Spermatorrhœa*, his *Manual of Hypodermic Medication*, and his four prize essays, of which one was on *Quinine*, one on the *Bromides*, and one on *Atropia*. In all of his occupations he accumulated material for the production of his well-known work on *Materia Medica and Therapeutics*, in which he garnered up his twenty-two years of experience as a practitioner. In this one volume he stated tersely, yet clearly, thousands of facts, pharmacological, physiological, and clinical, bearing upon therapeutics and diet. His avowed aim was to "stem the tide of therapeutic nihilism." It was immensely popular with doctors and students from the start, for the work of thinking was all done by the author, even to the prescription formulas. He had digested the current literature of the subject, weighed it by the light of his own experience and then set down his conclusions, which were usually optimistic. It is no exaggeration to say that his expressions of confidence in the powers of the materia medica had a marked influence upon the medical practice of this country. Bartholow's formulas, without doubt, added to the effectiveness of the energy that is in drugs. The widespread consideration they received is shown in the phenomenal circulation of eleven editions of the work, making a total of 60,000 copies.

In 1874 he published in the *American Journal of the Medical Sciences* a report which has a retrospective interest, and also illustrates his enterprise as the forerunner of all who operate on the human brain. His candid article has been republished by the anti-vivisectionists as a highly significant tract, and references to it are still seen in their literature. The circumstances are these: The valuable results flowing from the experiments made by Hitzig and Ferrier upon the functions of the brain in animals had made a decided impression upon those seeking data for greater certainty in therapeutics. No one had as yet made similar experiments upon the brain. A case came under Dr. Bartholow's care of rapidly extending epithelioma of the scalp with exposure of the dura mater. To the ardent investigator this was a timely opportunity provided by nature, and not to make use of it for extending the bounds of knowledge would be to fail in his duty. With the full consent of

the hopeless patient, who knew that life must soon be extinguished by the spread of the cancer, electric stimulation was applied directly to the posterior lobes of the cerebrum by needle electrodes. The results were confirmatory of those obtained in the lower animals by Hitzig and Ferrier, but at the time of the experiments, and for some hours after, there were complications which denoted that the knowledge was gained at the expense of some injury to the brain. The patient's death some days later was ascribed to extension of the cancer producing a thrombus of the longitudinal sinus. Dr. Bartholow got little credit for his daring in invading the sacred organ or his candor in reporting the whole affair. He was censured by medical journals at home and abroad. To his critics he replied that he had reason to expect that the faradic current would not prove electrolytic and that the tissues would escape damage; that the celebrated case of recovery after a crowbar had passed through the brain had been considered as proof that the brain was tolerant of injury, but that his recent case having proved the contrary it would be criminal to repeat such experiments. The editor of the *British Medical Journal* expressed the opinion that the apology of Dr. Bartholow disarmed further criticism. Since that time the whole science of cerebral surgery has been developed upon the assumption that the antiseptic method gives plenary indulgence for that form of sacrilege. Notwithstanding the comments of his censors, thought at the time to be damaging to his reputation, his rare powers of discrimination as a therapeutist eventually met with the recognition which they deserved. He received the degree of Doctor of Laws from Mount St. Mary's College and became a fellow of the College of Physicians of Philadelphia, a member of the American Philosophical Society, and an honorary member of the Royal Medical Society of Edinburgh and the Society of Practical Medicine in Paris.

About this time he was chosen for the vacant professorship of Practice of Medicine in the Medical College of Ohio, which he filled ably and acceptably for five years. Upon the death of Dr. John Biddle, the Jefferson Medical College elected Dr. Bartholow as his successor in the chair of Materia Medica and Therapeutics. This was in 1879, when he was forty-eight years old, with much the

"largest and most lucrative practice ever attained in Cincinnati." Even after his removal to Philadelphia many of his old friends and clients from Southern and Western States evinced their confidence and esteem by coming long distances to consult him. His health was not of the strongest, and yet the demands of his practice in Cincinnati had become almost ceaseless. He was tempted to leave these harassing cares by the prospect of leisure for a few years to finish his scholarly work on the *Practice of Medicine*, with which effort he hoped to crown his career. There could be no doubt of his ultimate success in his new field of labor. From the good fight he had fought in Cincinnati he had come off victor. By dint of his capacity for hard work, he might reasonably hope to win his reward anywhere, even on this difficult ground already occupied by medical men of the highest calibre. He brought to Jefferson Medical College the method of therapeutic study by experiments on the lower animals. Sharing the medical clinic with the late Prof. J. M. Da Costa, by his extraordinary knowledge of the resources of therapeutics, he fitly supplemented that remarkable diagnostician. His lectures were distinguished by conciseness and lucidity of statement and gave an impression of keen perception joined to energy and decision. His delivery was easy and fluent, his choice of words doing full justice to the breadth of his information and the precision of his thought. He made good use of his time of waiting for practice, and in the year following his removal to Philadelphia he brought out his *Treatise on the Practice of Medicine*, in which he incorporated the matter of his course of lectures on that subject at the Medical College of Ohio. It took at once a high place among books of intrinsic merit and, like all his publications, has won the approbation of many readers. The second edition was issued in three months and five other editions have appeared since. It has had the rare distinction of translation into the Japanese. In 1881 he delivered the Cartwright lectures on "The Antagonism between Medicines and between Medicines and Diseases." In Cincinnati he had founded the first weekly medical journal in the West, and when the *Medical News* of Philadelphia was reorganized as a weekly paper in 1882, he was one of the original members of the editorial staff, with his customary ability presiding over the department of Med-

icine, Therapeutics, and Medical Jurisprudence. His widespread reputation eventually brought him a large office consultation practice, which, with his attendance at the Philadelphia and Jefferson College Hospitals, his college and literary labors, kept him sufficiently employed. Leading the scholar's life in Philadelphia and making his summer home on Buzzard's Bay, Massachusetts, he had reason for the satisfaction he expressed to the writer when he stated that he would be content if he never crossed the Alleghanies again.

For a few years he filled the position of Dean of the Faculty, but his growing business made his official duties irksome and he resigned them in 1887.

In 1893, at the height of his renown, he retired from college work and was made Emeritus Professor. He had sustained a mental breakdown which necessitated absolute rest for a long period. His explanation of the attack was that some years before he had suffered from diabetes and to cure it had been put upon a course of opiates which he had seconded with other drugs. These had enabled him to go on with his many employments until he reached the limit of his endurance.

After his recovery, while he never attained to his old-time vigor, he resumed his practice, revised new editions of his books, and made numerous contributions to medical periodicals.

In his personal appearance there was an air of distinction due in part to his dignified demeanor and his careful dress. Of medium height and weight, his bearing was reserved and lacking in geniality. While his professional expertness was of undoubted value to society, as an unremitting student, with the habit of seclusion, the social life had few charms for him.

His cool and somewhat cynical manner gave no intimation of his alertness and mobility. With mental powers always in light marching order, he was ready for lecture, consultation, post-mortem, or critique, able to cope with any adversary that his aggressive energies might arouse. The secret of his material success won without the arts that make for popularity must be found in his mental capacity, industry, and resolution, qualities which united in one brain always give to the possessor ascendancy over others.

After a lingering illness he died at his home in Philadelphia,

May 10, 1904, aged seventy-two. There is a degree of satisfaction to his fellow-students of this body in the certainty that the best part of his work has found a place in the sum of knowledge available for curing disease, and that the inspiration of his teaching still lives in the precepts of his books and in the practice of thousands of his students.

ANNUAL ADDRESS OF THE PRESIDENT.[1]

By HORATIO C. WOOD, M.D., LL.D.

Gentlemen: Two years ago, on a distant Continent, an invalid was slowly casting behind him weary days amidst desert sands. This invalid you selected to be your President, although there was for him little hope of service. Great must have been the affectionate regard and sympathy that led you to confer the highest honor in your command upon such as he was. To-night he leaves the Presidential chair, and in so doing desires first of all to express to you his full appreciation of the great kindness you have shown him.

Two years have passed by. As is usual in this world, the unexpected has happened, and for the last eighteen months your President has been in all faithfulness able to fulfil, with whatever of ability has been given him, the duties of his high office. In the history of the College these eighteen months have been pregnant with events—happenings which to some of you have seemed sudden, and it may be even unnecessary.

Like as the century plant grows through many decades to unfold its blossom in a night, so is the usual history of development. In the present instance the changes which have seemed so abrupt have been the outcome of years of prosperous out-reaching. Decade after decade the College Library and, at a less rate, but still steadily, the College Museum have grown until, as old Rome has disappeared under its own débris, there has been

[1] Read January 6, 1904.

danger that the College would become but a disordered heap of uncared-for books and specimens.

Almost immediately after entering upon the active performance of duty your President became so strongly impressed with the imperativeness of the needs of the College for more space that he felt failure on his part to act would be dereliction of duty. The subject had been reported upon by several committees and discussed upon the floor of the College, but no exhaustive study of the situation had been made, and no practical remedy had been found. As the result of a very careful going over of the whole matter, the Presidential Address of 1903 was given to the College, and by order of the Fellows printed and distributed for general information.

At this point it may be allowable to say that in the preparation of the address the subject was approached with no preconceived ideas as to what ought to be done. For nearly forty years your President had been going in and out of the old College building; around it centred all the associations of his early and later medical life, and very naturally his inclination was in favor of developing the old habitation into something that would suffice for the new growth. Further, there was no thought in the mind of your President to impress his own views upon the College; indeed, at the time of the making of the address the Presidential opinion was unformed. The only attempt was to put before the Fellows of the College the facts of the case in as straightforward and simple a manner as possible. Investigation and discussion soon led, however, to crystallization; the logic of the situation was so strong as to quickly force decision.

As with the President, so with the Fellows. At first the general desire was strongly for the College to remain where it had so long dwelt, yet with the continuous discussion more and more of the Fellows were driven to the conclusion that the demands of the future required a larger space than is now occupied, until finally, at perhaps the largest meeting of the College ever held, by a two-thirds vote, it was decided to purchase the lot on Twenty-second street above Chestnut. The wisdom of

the choice has been justified by the result. The College holds in its possession the only large lot in the central portion of the city at all available, a lot which twenty years from now will be more accessible to the Fellows of the College than is the present site, a lot sufficient in size for the wants of the College for centuries, a lot to which there seems only one objection, namely, the smoke which at times is carried in its direction. This nuisance can, however, and should be largely abated. Not simply from motives of self-interest, but for higher reasons, the College should appoint a committee to assist in the present movement to abate the smoke nuisance in Philadelphia, so that the City of Homes shall not become a Stygian pit of darkness.

The next duty of the College is so plainly to go forward as rapidly as is possible and improve the lot that no discussion is necessary. It is only a question as to the ways and means of so doing; $150,000 has been offered and refused for the present building of the College; it clearly may be valued at $160,000. There is left of the Building Fund about $20,000; the Permanent Fund of the College, amounting also to about $20,000, is certainly available, making in sight a total of $200,000, which may be expended in building. The exact cost of the proposed hall cannot be known until the Committee on Plans has worked out some definite result, but an approximate working hypothesis can readily be formed. For the present purpose it suffices to suppose that a building 110 feet of frontage, 100 feet of depth, 70 feet of height—nearly twice the size of the one we now occupy—would suffice for the needs of the College for the next fifty years; and if properly planned, at the end of that or any other period, could readily be enlarged without unnecessary expense to meet any possible growth. A building of the size mentioned would contain 770,000 cubic feet. The so-called Arcade Building of the Pennsylvania Railroad, Fifteenth Street above Chestnut, cost thirty-one cents a cubic foot, and thirty cents a cubic foot may be taken as a fairly approximate estimate of what the College should spend on its new hall.

The history of the College would best be epitomized in the

Colonial style of architecture; adopting this style, and using brick in accordance with its traditions, for thirty cents a cubic foot could be erected a fire-proof building, simple but dignified in its exterior, handsomely and even luxuriously finished in its interior; 770,000 multiplied by 0.30 gives the sum of $231,000: allowing $4000 for the cost of moving and having $200,000 in sight, there is a deficit of about $35,000 for which the College must provide.

An analysis of the results of last year's efforts for the obtaining of subscriptions seems to your President to show that it will be comparatively easy to raise $25,000, and that $40,000 is not impossible. Of the $105,000 which was subscribed last winter, $76,530 was given by persons not Fellows of the College. Of this sum only $15,420 was contributed by non-Fellows living in Philadelphia. Certainly it is possible, or will be when Asphalt and Lake Superior are like past visits to Monte Carlo—simply half-forgotten bad dreams—to raise $20,000 from the general citizens of Philadelphia. To do this, however, it is essential for the College to be more liberal in regard to its Library. In the opinion of your President it would be suicidal to allow the general public to use the Library of the College of Physicians as a circulating library; but, on the other hand, it is Asiatic to surround this great collection of books with a high wall. It should be made a By-law of the College, that the Library of the College is to be open without charge six days in the week to anyone who wishes to consult it within the building. If this be done much money can certainly be obtained from the laity. It is, however, for this end essential that every Fellow of the College feel that he is a member of the Collecting Committee; very few of us but have some friend or patient who would give $20, $50, $100 or more, simply for the asking, and give it gladly.

Of the $105,000 raised, the Fellows of the College themselves contributed $28,470. There are at present some 326 Fellows, of whom only 198 have made any subscription to the Building Fund, so that not much over one-half of the Fellows have as yet

given. Nor is it the junior members of the College only who have been stricken with this forgetfulness; many who are most able to give have not yet put their names upon the list. The subscriptions vary from $10 to $5000; the minimum amount is so small that there is scarcely a Fellow of the College who cannot afford without sacrifice to contribute his mite; the range of the subscription is so great that every Fellow has opportunity to give according to his means. Certainly a considerable sum should be given by those who have not yet subscribed. Speaking for himself, and speaking from knowledge due to conversation with other Fellows, your President is assured that there are Fellows who have already subscribed who will give as much more as they have given, provided that a sufficient amount of money be raised to make it rationally sure that the new hall can be erected free from debt. It is only necessary that all the Fellows of the College, and not a mere majority, contribute according to their means, and that each Fellow shall resolve to obtain at least one subscription from a member of the outside public, and the money will be easily raised. For complete success but one thing is necessary: enthusiasm—an enthusiasm so virulent that it will infect the whole mass. Who will furnish the microbe?

A SMALL HOSPITAL EPIDEMIC OF PNEUMOCOCCUS INFECTIONS.

With Remarks on the Transmission of Pneumonia and the Methods by Which it Can Be Prevented.[1]

By DAVID L. EDSALL, M.D., and ALBERT A. GHRISKEY, M.D.

The extensive prevalence of pneumonia and the very high mortality of the disease have, within the last few years, been the subjects of frequent discussion; and there is general interest in determining the manner in which the disease is propagated and the methods by which it can be controlled. We know that pneumonia is an infectious disease, and we know many facts concerning the bacteria that usually produce it. We recognize also that infections do not arise from mysterious sources that are essentially beyond the human ken, but each day see more clearly the fact that those sick with infectious diseases constitute the supremely important sources of dissemination of those diseases. We are likewise growing into a clearer recognition of the equally important fact that bacterial diseases are not in part transmissible, in part intransmissible, but that they differ only in the readiness and directness with which they may be transmitted.

Yet the attitude of the profession toward the control of pneumonia has been somewhat passive; for in spite of this general knowledge there are certainly few physicians who adopt more active precautions to prevent the spread of the disease than to have the sputum from pneumonia patients destroyed, and perhaps to utter a gentle warning as to the possible danger in very close contact with those sick of the disease.

[1] Read January 6, 1904.

It is quite true that direct transmission of pneumonia from case to case seems to be comparatively rare; that the disease does not generally appear in epidemic form, but develops sporadically; and that the immediate sources of infection are, at present, usually not discernible. But these facts do not, in our belief, justify the statements recently made by a number of authors that until our knowledge of the manner in which the disease is transmitted becomes more exact we cannot hope to exercise much control over the spread of pneumonia; and should not urge the general public to adopt measures for avoiding the disease, lest such suggestions cause undue apprehension. As a rule, we are unable to protect individuals from pneumonia, and can only urge upon them general hygienic measures, such as those recently described by Reynolds. We can, however, by giving the public a proper understanding of the matter, probably prevent a large proportion of the cases of direct transmission; we can, in localized epidemics, search for the source of infection, and sometimes discover and destroy it; and we can with wisdom undertake somewhat more active measures than those usually adopted to prevent the spread of the infection from its chief source—*i. e.*, from those that have acquired the disease.

We are, to be sure, limited to general principles in carrying out most of these measures; since we cannot follow the course of the infectious material so clearly as we often can, for example, in those infectious diseases, such as typhoid fever, that are usually water-borne. It is, however, scarcely to be expected that special methods of preventing each individual infection, particularly those that are air-borne, will be discovered for many years, if at all; consequently, for a long time to come, and perhaps for all time, our only method of controlling most infections will be through the use of the general principles of preventive medicine.

That such methods are effectual, although the work slowly has become apparent through the results in tuberculosis, which furnish an inspiring illustration of the possibilities in combating a disease that is largely air-borne, when it is fought by a widespread attempt to institute general hygienic measures and by arousing general public interest. Tuberculosis is not the only disease that will respond to such measures. It has become, rather, as Clifford Allbutt puts it,

"the flogging schoolmaster" driving in facts concerning the control of infections; and many of the principles that are used in preventing tuberculosis are applicable in controlling pneumonia. This is indicated by our general knowledge of the disease, and particularly by the literature on the subject of epidemic pneumonia, to some of which we shall refer. The fact that these measures may be less successful in pneumonia than in tuberculosis is no argument against their adoption if they are likely to meet with any success at all.

We shall first report a series of recent observations of our own. While the number of cases is small they exhibited some points that are of importance in relation to epidemic pneumonia.

When one of us (Edsall) went on duty in the men's medical wards of the Episcopal Hospital, April 1, 1903, there was, in the small side ward, which contains six beds, a case of extremely grave pneumonia, the patient occupying bed 2. This man was at that time but half conscious and was completely prostrated. He had extensive consolidation of both lungs, and also arthritis of the right wrist-joint. He was evidently overwhelmed by pneumococci; for blood cultures gave a free growth of these organisms, not only when large quantities of blood were used, but even with tube cultures on hydrocele fluid, and when only two drops of blood were used.[1]

It is worth while, in view of subsequent developments, to note that on account of the patient's mental condition it was impossible to prevent his expectorating on the bedclothes and the floor. The next day he was still worse, and on the day after (April 3d) he died. The bedclothes were, of course, entirely changed; the bed was aired, as usual; and the neighboring portion of the ward was repeatedly cleansed, both during and after the man's illness.

On April 4th a man in the second week of a mild attack of typhoid fever was admitted and put in this bed. When he had been in the bed about twenty-four hours blood cultures were taken for the typhoid bacillus. To our surprise the culture showed a pure growth of pneumococci. This patient exhibited no leukocytosis, either at this time or at any subsequent period; and he had no signs of pneumonia or of any other local pneumococcic infection, except a fairly

[1] This and the other bacteriological work reported in this paper was done by Dr. Ghriskey.

marked bronchitis, which might possibly have been of this nature. The pneumococcus could not be looked for in the sputum, as he did not expectorate. The bronchitis persisted for ten days, but did not influence his general condition. He passed through a mild attack of typhoid fever and recovered entirely.

This man had meanwhile (April 5th) been transferred from the previously mentioned bed in the side ward to the main ward. On April 6th a man of twenty-two years with sciatica was admitted. Because his suffering from the sciatica led him to be extremely noisy he was placed in the side ward, and chanced to be put in bed 2. He remained there for about thirty-six hours, and was then transferred to the main ward. Less than two days after this transfer had been made he developed pneumonia of the entire left lower lobe, which, however, was of mild and very brief course. The patient showed but little evidence of toxæmia except fever, which ran to about 103° F., crisis occurred on the third day, and undisturbed resolution proceeded rapidly from this time.

On April 8th a boy aged fourteen years, with tubercular meningitis was placed in bed 2 in the side ward. He had previously been in bed 5 in the same ward for a week, and during that time had exhibited the ordinary symptoms of tubercular meningitis, with leukocytosis, and without any signs of pulmonary involvement. Two days after having been placed in bed 2 he showed an increase in temperature, which persisted; he developed a leukocytosis of 14,000; and his respirations grew rapid, and remained so. He was already stupid and was breathing poorly, and it was difficult to make a satisfactory examination. It was never determined that he had pneumonia; but we have always suspected, in view of the facts concerning the other cases described in this report, that he had either pneumonia or possibly some other form of pneumococcic infection. Five days after having been placed in this bed he died. A postmortem could not be obtained.

On April 14th, the day after this patient's death, a man aged forty years, with mild alcoholism was placed in bed 2 in the side ward. On the following day this man was apparently in almost normal condition, except that he was somewhat exhausted. On the 16th he exhibited a sudden rise in temperature to 102° F., and had pain in the right

side; and later in the day he developed clear signs of pneumonia of the right lower lobe. These symptoms continued for two and a half days, with otherwise very mild constitutional disturbances. There then occurred a sharp crisis; the pulmonary exudate underwent rapid resolution; and the man proceeded to a perfect recovery, though his convalescence was much prolonged by persistent and exhausting diarrhœa.

On April 15th there was admitted and placed in bed 5 in the side ward (opposite to and about ten feet away from bed 2) a man at the end of the first week of typhoid fever. On the 17th a blood culture was taken from him with the expectation of finding the typhoid bacillus. Again a pure culture of the pneumococcus was obtained. This, man, like the other typhoid patient, ran through a mild course of typhoid fever of the usual duration. He exhibited no leukocytosis, and had not even a noteworthy degree of bronchitis, and there was no evidence of any other complication of his typhoid fever. Only one peculiar clinical feature of the case was noted: the temperature showed remarkable diurnal fluctuations, ranging nearly every day throughout the fastigium of the disease from normal or from slightly below or above normal to 102° F. or 103° F. He recovered without any special incidents in his case.

When the second case of pneumonia developed in bed 2, and when, to our great surprise a second pure culture of pneumococci was obtained in this ward from a typhoid patient, it appeared evident that a pneumococcus contagion was occurring. The ward was afterward formalized, and no cases occurred subsequently.

From the story that has been told it may seem that it was only carelessness or inattention that permitted these conditions to continue as long as they did. This, however, is hardly true, for the side ward is used largely for the temporary detention of patients who are noisy or are in other ways likely to cause disturbance in the main ward. Patients are usually kept in the side ward for a short time only, and are then transferred to the main ward; and of course there is, as a rule, no occasion to make a note of the beds in which they have been placed in the side ward.

At the time that these cases occurred the service was very active, as many as six or seven new cases being frequently admitted to the

men's wards in a day, and it was not at first evident that events had followed the course described.

The typhoid patient from whose blood pneumococci were first obtained had already been in the main ward two days when the result of the culture became known. The man with sciatica had been in another part of the main ward for two days when his pneumonia developed. There was then a pause of about a week, during which time nothing especially suspicious happened. At the end of this period the second case of pneumonia developed, and the second instance of pneumococci in the blood of a typhoid patient appeared. After this the possibility of contagion at once suggested itself and was acted upon, and investigation showed the facts that have been stated concerning the beds that the patients mentioned had occupied.

This little epidemic, then, consisted of only two cases of actual pneumonia, together with two extremely peculiar cases in which pneumococci were apparently present in the circulation without causing any of the usual signs of pneumococcus infection. In spite of the paucity of cases the observations are, however, interesting in several ways: in the unusually striking evidence that a well localized source of infection had been provided by the original fatal case; in the evidence concerning the period of incubation, and in the extreme mildness of the infections. In connection with the latter point, the two typhoid cases that yielded cultures of the pneumococcus are of special interest, though their meaning is perhaps somewhat questionable. They require particular discussion, which will be given later.

The fact that pneumonia sometimes becomes distinctly epidemic is freely granted by all who have interested themselves in the matter, and is now sufficiently well known to be the subject of frequent comment in the public press. The belief that the epidemicity of the disease is a recently established fact is not uncommonly met with, though it is hardly necessary to say that such a belief is entirely erroneous. During the last quarter of a century attention has been especially directed to this point, but the fact that the earlier literature contains numerous references to pneumonia epidemics may be seen at a glance by consulting Hirsh. This author, in discussing the history of the disease, collected massive evidence

of its epidemicity from the literature of the earlier part of the nineteenth century and from that of several preceding centuries; and many of the records to which he refers are unquestionably authentic. His reference to Savonarola seems to demonstrate that a belief in occasional epidemic outbreaks of pneumonia and apparently in the contagiousness of the disease existed at least as early as the middle of the fifteenth century.[1]

It is of interest that Savonarola, as well as numerous other early authors, mentions conditions not unlike those that existed last year in Chicago and this year in New York City. The terribly destructive epidemics of pneumonia in those cities have excited great public interest, and have been discussed by many of the laity, and indeed by many of the profession, as if they were unprecedented.

But while the epidemicity of pneumonia has long been recognized and is now well established, there has until recent years been a strong tendency to attribute epidemics in general, as well as the individual cases observed in these epidemics, exclusively to atmospheric or other indefinite source, and to ignore or flatly deny the possibility that those sick with the disease may transmit it to others. At times a voice was heard insisting upon the occasional contagiousness of pneumonia; but stronger voices held the ear of the profession. It is not at all remarkable, however, that in recent years the attitude concerning this point has undergone a decided change. The demonstration that pneumonia is an infectious disease, and that certain well known bacteria (especially, of course, the pneumococcus) are its usual causes, has opened the way for the ultimate recognition of its possible contagiousness; and with the advance in the knowledge of infections, the rigid but artificial distinctions between contagion and infection that were once made have been gradually cast aside, until now there is no distinction between the two terms that can be constantly maintained—unless it be based upon necessity for residence of the causative agent in an intermediate host.

The literature on epidemic pneumonia makes it practically clear

[1] Quoted by Hirsh, Handbuch der Historisch.-Geographisch. Pathologie, Bd. iii., p. 125; Savonarola (Practica, Tr. vi. cap. 10, rubr. 13, Venet., 1497, p. 113 b): "In civitate nostra Paduæ et Trevisii et Venetiæ 1440 de mense Martii autea et post apparuit pleuresis mala quæ erat contagiosa, ex qua multi et plurimi moriebantur."

that infection may occur (1) through widespread atmospheric dissemination; (2) through the agency of some local source; and (3) through direct contagion from persons sick of the disease.

A review of the literature of the last three decades bearing upon the contagiousness of pneumonia can scarcely leave any one unconvinced of the fact that direct transmission sometimes occurs. It is quite true that absolute proof of this is exceedingly difficult to obtain, but the circumstantial evidence is so strong and has been so frequently furnished that it can no longer be rationally denied. A number of authors and many practitioners seem to be still skeptical concerning this point. They may properly remember, however, that even in the diseases in which direct contagion is known to be the common method of transmission we cannot, in a vast proportion of cases, demonstrate that this has occurred or determine the source of infection, and that in tuberculosis, for example, absolutely indubitable evidence of the source of the disease is almost impossible to secure; yet men of rational thought scarcely deny, nowadays, that case-to-case transmission of tuberculosis frequently occurs.

The chief evidence that contagion takes place in pneumonia is afforded by a large number of observations of instances in which several members of a household or other persons that had come closely into contact with each other have sickened with this disease soon after the development of a primary case among their number. In some of these instances the activity of indirect atmospheric infection was made extremely improbable by the fact that persons living in a region in which pneumonia was more or less epidemic have escaped the disease for a considerable length of time, until they had either visited or assumed attendance upon a pneumonia patient; but that soon after exposure they had been taken ill with the disease. In a number of these instances the person or persons who acquired the disease slept in the same room or in the bed with the original patient.

A number of broad general facts indicating the contagiousness of pneumonia have been brought out by Riesell: (1) As just stated, there are, in epidemics of the disease, many instances of escape from attack prior to visiting infected houses or to actual contact with other cases, while attack has followed such exposure. (2) In epidemics

the disease is often confined to one settlement, and the cases occur chiefly among those coming into close relations with one another; while neighboring regions, in which the conditions of ground-soil, etc., are entirely similar, escape. (3) Usually there is, at the same time, a widespread distribution of the cases in a settlement that is actually infected; this, in connection with the last point, indicating transmission from case to case, rather than local soil-infection or the like. (4) In connection with the latter point, Riesell mentions also the rarity of absolutely isolated house epidemics; and, further (5), the time periods between the appearance of individual epidemics in small, isolated villages make improbable the view that these epidemics are always due to conditions of the atmosphere or of the soil. Riesell considers that these conditions cannot be entirely explained by peculiarities of the ground-soil or of the atmosphere, but that they can be explained upon the basis of transmission from person to person.

There are so many observations of instances in which contagion seems extremely probable, and this question is so thoroughly settled, that it is not advantageous to attempt to discuss the literature concerning it. A recent observation made by Spaet deserves, however, a little attention. In the epidemic at Neubrunn, which lasted from the early part of May to the middle of July, 1903, there were sixty-three cases in a population of 454, and over fifty of these cases occurred prior to June 6. Of the remaining cases, five developed within three days (from June 22 to June 24, inclusive); and all these five cases occurred in persons who had attended the funeral of a pneumonia patient on June 12, the funeral having been held in the infected house. While these facts do not demonstrate positively that these five persons had become infected in the house mentioned, the observation is of interest in its suggestion that a very slight exposure may cause infection.

The circumstances attending hospital epidemics of pneumonia offer some of the most striking evidences of its occasional contagiousness. That hospital epidemics occur has been clearly recognized for years; and since these epidemics are usually limited to one ward, the probability that contagion spreads the disease is always extremely great. Indeed, this has, in many cases, been almost absolutely proved.

The cases reported by us are important in that they show with remarkable clearness the evidences of local infection derived from the original case.

We have referred to the fact that many epidemics of pneumonia are not due to direct infection from another patient or to a traceable intermediate infection—indeed, the contrary is naturally much more frequently the case. In widespread epidemics, in which a great many cases occur over vast areas of country, we are obliged to believe that the cause is atmospheric infection, like that which seems usually to produce epidemics of influenza; and the source of infection in such instances is, with our present methods, usually undiscoverable. There are also on record numerous instances of localized epidemics in which it is apparently quite clear that the infection has been atmospheric, coming from some untraceable source. An example of this is the epidemic at Ober Sitke described by v. Holwede and Münnick. In this village, with a population of about 400 persons, many cases occurred within a short time, chiefly among the children, of whom there were about fifty. Within thirteen days fifteen of these children sickened with pneumonia. At the time of this epidemic, owing to the extremely inclement weather, most of the children in the village had been kept housed; for this reason, and because many of those who became ill lived in widely separated houses, between which there had been no intercourse, it seems impossible that there could have been any question of contagion or of local soil-infection and the like.

In another class of instances evil sanitary conditions have been found in areas in which there were local epidemics; and this fact has furnished presumptive evidence that the bad sanitation was responsible for the outbreak, although no direct connection of the source discovered with any primary case or cases could be demonstrated.

An example of this is seen in the epidemic mentioned by Winter, which occurred in a localized portion of the town of Kappelrodeck, and persisted from November, 1890, to February, 1891. The chief interest in this epidemic lies in the fact that it occurred in a thickly populated alley, whose inhabitants were extremely poor and lived in particularly bad hygienic surroundings. The alley lay at the foot

of a steep granite hill, impermeable to moisture, upon which manure and other refuse were regularly spread. Typhoid epidemics were common also in this district; and it seemed probable that the epidemics of typhoid and perhaps the pneumonia epidemic could be referred to the situation of the alley in relation to the hill. There was certainly every opportunity for typhoid infection to have occurred through infection of the drinking-water from the refuse-laden hill.

Instances of a character intermediate between that last mentioned and direct contagion—*i. e.*, instances in which there was evidence of soil-infection, but in which it was fairly clear that the soil-infection had come from earlier cases—are on record, as, for example, that of Chrostowski and Jakowski. These authors describe an epidemic that involved six persons. It occurred in a bakery, and was apparently due to the fact that, on account of imperfect plumbing, a temporary closet with an open trench had for some time been in use in this house. It was subsequently learned that for years past isolated cases of pneumonia had been regularly received each year from this house in neighboring hospitals. The soil had probably become infected from the earlier cases: and when the trench was dug the organisms had reached the air directly, and had thus caused the epidemic. The authors state that they made bacteriological examinations of the soil and animal inoculations, and demonstrated the presence of the Friedländer bacillus; and the same organism was shown in two of the cases by aspiration of the lung. A similar observation is that made at the Amberg prison, where there had been several severe epidemics of pneumonia, and where each year numerous cases of pneumonia appeared among the prisoners. The soil there also was shown to contain large numbers of the Friedländer bacillus. Whether this organism was the cause of the cases of pneumonia or not, there can be little doubt that there was a local infection that had produced the repeated outbreaks, and had likewise caused the undue number of cases each year.

We would mention also a similar and very suggestive observation that was made by Tamforde in the epidemic that he describes, the history of which is, in brief, as follows: A man sickened with pneumonia in September, 1901. He lived in a small house in very restricted circumstances. He expectorated on sand, and his wife

regularly emptied this sputum-laden sand on the manure-heap. The man recovered, and soon afterward the manure was spread over the neighboring fields, which were at that time flooded and frozen over. Mild weather prevailed during December and the early part of January. During the latter part of January and the early part of February strong winds blew most of the time from the west and southwest, and during this period a definite epidemic developed, almost all the cases occurring in persons who lived in the vicinity of the infected fields, and practically exclusively in those living to the east of those fields—*i. e.*, in the direct path of the wind coming from the infected area. The previously mentioned man who had apparently produced this epidemic himself fell ill again with pneumonia. The course of events in this instance is too impressive to need comment.

The point of chief importance that is demonstrated by the literature on epidemic pneumonia is one already known, but not sufficiently well recognized, viz., that while we are limited to general hygienic measures in controlling extensive atmospheric infection, local epidemics may often be stamped out by making a proper search for their cause: and when direct transmission is active in producing the disease, we can frequently stop its progress at once by instituting sufficiently active measures.

Pneumonia, however, does not usually tend to become epidemic, and demonstrable contagion certainly occurs with comparative rarity. The point that is at present of chief clinical interest is to determine, as far as possible, the reasons for the occasional greater epidemicity of the disease, and for its occasional tendency to produce contagion. An insufficient amount of study has been given to this point, especially by clinicians. Netter, Riesell, and others refer to the fact that actual contagion seems to occur chiefly when the contact is intimate, and that the danger of contagion is considerable only in the cases of those who are in attendance upon pneumonia patients or occupy beds near them. This usually seems to be true, but not always, as contrary observations have been made. Contagion is also, of course, dependent to a considerable extent upon the presence of other infections, particularly influenza. It is well known that pneumonia becomes especially prevalent during influenza epidemics, and the literature also demonstrates that evidences of direct contagion from

pneumonia cases have been noted with especial frequency at such times. The latter point was strikingly illustrated in the ward epidemic of Lancereaux and Besancon, which involved four ward patients, an attendant, and an externe. The six cases of ward infection, all of which were fatal, occurred within a few days of one another, and at a period when grippe was epidemic; and several of those who acquired the disease either had actual grippe or had been in attendance upon persons with that disease.

Another possibility that may be briefly mentioned is that suggested by Dieudonné, who notes that epidemic pneumonia has repeatedly been observed in human subjects at the same time that epidemic pneumonia has been prevalent among horses, and that many of those having the care of the sick animals have acquired the disease. It is wholly possible that pneumonia may occasionally be transmitted from horses or other animals to man.

Most important subjects of investigation, however, are those relating to the virulence of the organisms producing the disease and to the viability of these organisms in different media and under various conditions. Owing to the older teaching of bacteriologists, the view is prevalent among clinicians that the pneumococcus rapidly loses its virulence and dies out in the sputum of pneumonia patients and in culture media. A series of investigations made within the last ten years have, however, shown that virulent pneumococci may at times persist for a considerable period in the sputum of convalescents from pneumonia (Bignami and Marchiafava, and others); and it has also been demonstrated that the organisms may long remain viable and virulent in preserved sputum (Foa and Bordoni-Uffreduzzi), and in their natural media—dust, earth, etc. (Germano). Bordoni-Uffreduzzi particularly tested the persistence of the virulence of the pneumococcus in sputum, collecting the sputum on linen and drying it, partly in diffuse daylight and partly in direct sunlight. As was to have been expected, the virulence persisted much longer when the sputum was not exposed to direct sunlight. Under such conditions it was marked even after forty-five days.

Germano carried out an important study concerning the viability of the pneumococcus in various natural media. He found that when

mixed with dust, sand, and earth, the pneumococcus exhibits a varying degree of viability, depending to some extent upon the medium. One variety died rapidly, while another lived as long as forty days in dust and sixty days in fine sand. When blood containing the latter diplococcus was mixed with sand, or when similar experiments were carried out with pus containing diplococci or with sputum containing these organisms, living, virulent diplococci were recovered as long as one hundred and forty days afterward. Germano agrees with Kruse and Pansine in referring the discordant results of various previous investigators to differences in the variety of the organisms studied and in the media used. He believes that he has clearly demonstrated that a pneumococcus planted in its natural media may be found to be still living after nearly six months. It apparently retains its virulence even better when dry than when moist—a matter of evident importance in connection with air infection. Low temperatures are somewhat unfavorable to its growth, but not by any means absolutely so. Chantemesse made similar observations, and attributes the greater resistance of the organism when in such media to the fact that it is then enclosed in an albuminous mass, and is thus protected from absolute drying. Chantemesse refers to a hospital epidemic of pneumonia that was apparently controlled by taking care that no dust was stirred up and that the floor was daily treated with disinfectants.

Germano observed that a variety of pneumococcus midway between a diplococcus and a streptococcus in its characteristics showed particularly marked persistence of viability as well as of virulence. That organisms with such characteristics are usually virulent has been noted by others. Germano's organism was isolated from a case of pneumonia that occurred during a very severe epidemic of the disease. There was good reason, in consideration of his results in this instance, to believe that this special epidemic was due to the great resistance of this diplococcus and to its dissemination through the air.

The observations mentioned are of great importance in relation to the hygiene of pneumonia cases, since they demonstrate very clearly the importance of infection of soil, dust, etc., in disseminating the disease. They also furnish experimental evidence of the fact,

already made reasonably apparent by the clinical observations quoted, that a local source of infection may persist for a long time, and needs to be looked for, and if found, destroyed; and they offer an explanation of the occasional persistent occurrence of pneumonia in hospital wards and in certain houses, and likewise give us a clearer conception of the possibilities of contagion. They demonstrate extremely forcibly, too, that we may apply to pneumonia Koch's recent emphatic statement concerning the prevention of typhoid fever—*i. e.*, that success depends chiefly upon the rigid care in keeping the infection from spreading beyond those sick of the disease, not in devoting attention to the almost hopeless task of attempting to control an infection that is already scattered broadcast.

While we have no new or startling recommendations to make, we do believe that it is possible, without unduly exciting the laity, and without demanding an unreasonable amount of trouble and expense, to do more than is commonly undertaken to control the spread of the disease. There is no doubt that some instances of direct transmission could be prevented by educating the laity in general to understand that there is some danger of acquiring the disease from those ill of it. The fact that the danger is usually slight does not justify one in ignoring it; and it is not necessary to cause excitement in uttering a warning.

Reynolds goes so far as to recommend the use of measures of control similar to those adopted in cases of diphtheria. It seems to us that the strict isolation and disinfection practised in that disease are at present scarcely feasible in pneumonia. Nevertheless, we consider it to be wholly practicable to maintain a fair degree of isolation and to insist that those in ill health, especially if they have catarrhal processes of the respiratory tract, influenza, or similar conditions, shall not attend upon the patient or visit his room. The same rule should apply to all children, whether sick or well.

In hospital work, the ideal—but at present almost impracticable—procedure would undoubtedly be to isolate cases of pneumonia in a separate ward. As previously stated, however, the evidences of direct transmission seem to have been observed chiefly when the contact has been comparatively close; therefore, as Netter has suggested, it would perhaps be sufficient, and it would certainly be wise,

in hospital wards to separate pneumonia cases as far as possible from other patients; and one should insist emphatically that they be well separated from persons with influenza, typhoid fever, and other depressing acute diseases.

It is customary among well-trained physicians to have the sputum from pneumonia patients carefully collected and destroyed; but this custom is by no means general, and its importance may wisely be forced further upon the profession as well as upon the laity. Even, however, when this measure is properly carried out it is unquestionably insufficient. Many investigations during the past few years have taught us that tubercle bacilli are scattered about freely by tuberculous patients in coughing, sneezing, etc.; and the same thing has been shown to occur with other bacteria, and undoubtedly occurs in pneumonia. Even more important than this is the fact that it is often absolutely impossible to collect sputum carefully, since the patient is frequently delirious or half conscious, and the bedclothes and the floor are constantly being infected. The only satisfactory procedure is to have the bedclothes boiled, and the furniture and the floor thoroughly and frequently cleansed. If possible, in private practice the room should at least be kept well aired and closed off from the rest of the house for some time after the disease is over before being reoccupied by the patient or used by any other person.

We strongly suspect that the future will see the custom of actually disinfecting after almost every bacterial disease. At the present time, if evidence of direct transmission of pneumonia appear in a house or in a hospital ward, it is certainly incumbent upon the attendant to undertake actual disinfection as soon as possible. If there is any evidence that a local source of infection is producing a local epidemic, or if an unusual number of cases have occurred from year to year in a local area, it is also incumbent upon the attending physician to attempt to discover the source of this infection and to make efforts to destroy it.

The control of the disease would also be largely aided if it were required that pneumonia be reported to health boards. It is evident that this will soon be done in some of our large cities, and it unquestionably should be done generally.

If measures such as those that we have sketched were carried out persistently in hospitals and in a considerable proportion of private cases, and if at the same time public interest in the use of general hygienic measures such as those recommended by Reynolds and other health officials were widely aroused, we do not believe that such pessimistic views as those that have recently been repeatedly expressed in regard to the influence of hygiene on pneumonia could long be maintained. Any control over the disease that we may be able to exercise will necessarily come slowly, but we certainly shall not acquire much control until we adopt more active and more detailed measures than those we now employ.

We have referred to the mildness of the infection in our cases. It is frequently stated by writers on pneumonia epidemics that the disease when readily transmitted runs a grave course, exhibiting many nervous symptoms, great prostration, often enlargement of the spleen, and other evidences of severe infection. This is usually true, but not constantly, for various authors have described epidemics in which mild cases were common. Spact, for example, mentions a number of cases in which the crisis occurred by the fifth day; and Osthoff refers to a number of cases of abortive form which lasted three days or less. In our two pneumonia cases the course was very mild and extremely limited. the longer case having had its crisis at the end of three and a half days, and the other having ended by crisis in a little less than three days. If the two other cases be admitted as pneumococcic infections, they must be considered to have been extremely mild, having produced practically no symptoms or discoverable signs of local disease. This fact is of interest, in that it demonstrates that a high degree of virulence is not an essential accompaniment of transmissibility; and it shows that transmission should not be suspected only when the cases are severe.

As we have also indicated, the observations are of interest in relation to the period of incubation. There are very few records that give any direct indication of the minimum length of the period of incubation—a fact that is natural, as it is impossible in most instances to state when the opportunity for infection has occurred. Our cases are of unusual importance in this regard, because they demonstrate with striking clearness the length of the period of

incubation, and show that it must have been less than forty-eight hours in one case, and only a little more than that length of time in the other. Hoffmann considers that the period of incubation is usually about nine days; but in an epidemic he observed cases in which the shortest incubation period was probably about three days, the maximum having probably been about twenty-five. Other authors —Waibell, for instance—have observed evidences that the period of incubation may be very brief. Its greatest possible length is, of course, exceedingly difficult to surmise.

The two cases that we have mentioned, in which pneumococci were discovered in the circulation without evidence of local lesion, deserve brief discussion. We have no doubt that the results of the cultures in these cases were entirely reliable, and see no reason to question the belief that these patients had pneumococci in the circulation. The blood was drawn with the utmost care as to asepsis, and if any accidental outside infection occurred, these were the only instances of it in a large series of blood cultures made by one of us (Ghriskey) in the same wards and at about the same time. The cultures were taken by adding 10 c.c. of blood to 300 or 400 c.c. of 6 per cent. glycerin bouillon. Subcultures were also made on agar, glycerin-agar, and hydrocele fluid. All the cultures were characteristic, and they leave no reason for doubting that the organisms were pneumococci.

These observations are not entirely without parallel. Prochaska has reported four cases in which there was no pneumonia and no evidence of other local pneumococcic infection, except bronchitis, and in one case hemorrhagic nephritis. One of these cases was fatal, and the absence of pneumonia was shown at autopsy. In all these cases pneumococci were found in the circulating blood. In two of the cases the bronchitis was slight. In all the cases, however, the symptoms were severe.

Recently, Baduel and Gargagno have described a more extensive and more pertinent observation, though the details are given with unsatisfactory brevity. The points of chief importance in their observation are as follows: Eleven members of one household were demonstrated to have pneumococci in their circulation. Some of these patients were seriously ill, and the cultures were made for that

reason; while in others the clinical symptoms were so slight (bronchitis, pharyngitis, etc.) that no especial attention would have been paid to these cases had it not been for the household epidemic of pneumococcic infection that was already known to be present. Because of this epidemic blood cultures were taken, and they showed pneumococci in the circulation in all the members of the household.

The profession has, chiefly through the work of Prochaska, been led to give up the idea that positive blood cultures of the pneumococci necessarily mean a grave prognosis. It is certainly questionable, however, whether we have even yet recognized the frequency with which pneumococci and other organisms enter the circulation and are recoverable by culture, particularly when there is very little general evidence of the disease. Most clinicians hold to the view that positive blood cultures, in cases of supposed general septicæmia, are confirmatory of the diagnosis of what is termed septicæmia, and also have extreme prognostic significance. It may be that this idea also will have to be greatly modified. It certainly will if the observations made by Bertelsmann are substantiated. This author has made a large series of blood cultures in cases of local surgical conditions, such as abscess, periostitis, etc. In forty-eight cases he found bacteria in the circulation; twenty of these patients died after exhibiting the symptoms of general septicæmia: but most of the twenty-eight showed no evidence of septicæmia, and in many instances there was nothing but what we are accustomed to consider definitely localized pyogenic infection. Such observations if confirmed must modify one's views concerning the clinical significance of positive blood cultures in various conditions; and they are also likely to have an interesting influence upon the question whether many infections, such as pneumonia, are first local and general, or *vice versa*, as well as upon a number of other points of importance in the study of infection and immunity.

It is worthy of note that in our two cases that are now under special discussion pneumococci were discovered through the purely accidental fact that we were at the time interested in making a series of cultures for typhoid bacilli, and thereby chanced to light upon the pneumococci; and both Prochaska's and, particularly, Baduel and Gargagno's cases were discovered solely through deliberate exami-

nation of a series of cases that seemed from the ordinary standpoint unlikely to yield pneumococci. This is of interest because bacteria are usually searched for in the circulation only when there is good reason to anticipate that they may be there.

It is important on both theoretical and practical grounds that we should gain more extensive and more exact knowledge as to the frequency with which, by modern methods of culture, bacteria of various kinds can be found in the circulation when clinical indications of their presence are absent, but especially when exposure to the possibility of infection has occurred. It is possible that bacteria may not infrequently enter the circulation in large numbers and be recoverable by culture, and then be rapidly killed off; hence special interest would attach to blood cultures taken soon after exposure. In both our cases the cultures happened to be taken within thirty-six hours of the first exposure.

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ADDITIONAL OBSERVATIONS UPON RETROPERITONEAL SARCOMA.[1]

By J. DUTTON STEELE, M.D.

During the four years that have elapsed since my first review of the literature of retroperitoneal sarcoma nothing new has been contributed to the diagnosis and symptomatology.

However, many new cases of great interest have been recorded in current literature. These cases have been studied with greater care than was formerly the custom to give to such tumors, especially with regard to the physical signs and the relation of the colon, small intestines, and stomach to the growth.

Witzel's observation upon the relation of the hollow viscera to tumors originating in the retroperitoneal space have been accepted as one of the most important diagnostic signs of this condition.

In general it may be said that the attention given to such tumors is beginning to assume the importance that they deserve, and the physical signs and symptomatology are recognized as forming a definite symptom complex.

Rudolf Goebell has contributed perhaps the most important article of the past four years upon solid tumors of the retroperitoneal space. He considers the subject of lateral retroperitoneal tumors of all sorts, and includes in his list thirty cases of sarcomata. His series contains ten cases to which I did not have access. The remaining twenty were included in my first series. He does not report any new instances of the condition, nor do his conclusions add anything new to the diagnosis or symptomatology. His paper

[1] Read March 2, 1904.

is classical in its thoroughness, but is confined to the consideration of lateral tumors, and is consequently limited in its scope.

M. L. Harris and M. Hersog have contributed an excellent article upon the tumors of the mesentery proper, that is, those that grow in or around the lymphatic glands situated between the layers of the mesentery. Such tumors have little in common with solid tumors of the retroperitoneal space; and none of the fifty-seven cases which they have collected are available for my list.

Howard J. Williams reports three new cases of retroperitoneal sarcoma, which are included in my second series. He states that he has collected eleven new cases; but on referring to the authorities given, it was discovered that nine of these were tumors originating in the mesenteric glands, and therefore cannot be considered retroperitoneal as the term is generally used. The tenth case (reported by R. C. Smith) was included in my second series, and the eleventh case to which he refers could not be found in the reference given.

Dr. Williams gives an exceedingly interesting review of the subject, especially the operative treatment of the condition. He has seen more cases of retroperitoneal sarcoma than have been reported by any one American observer.

Otto Hartman reports a new case of upper central retroperitoneal sarcoma which is referred to at length as Case 69 of the new series.

R. Douglas reports one new case and gives a short excellent review of the symptomatology and diagnosis of such tumors.

In addition new cases have been reported by W. E. Hughes, John McLachlan, R. C. Smith, and J. M. Withrow.

My first series contained sixty-one cases. The second series contains those that have been recorded in current literature since January, 1900, and those that were overlooked or to which I had no access at the time of writing my first paper, and amount to thirty-five in all, including three new cases of my own.

The first series, together with the second series and three new cases, amount in all to ninety-six cases, upon which the following conclusions are based.

Symptomatology. The onset of retroperitoneal sarcoma is insidious, and the symptoms are not characteristic until the tumor has become large enough to be detected by physical examination. The

growth generally manifests itself first by signs of pressure upon some of the structures in the peritoneal cavity or retroperitoneal space. The character of these symptoms can best be described by considering the results of pressure produced in the different regions of the abdomen.

In tumors developing in the upper central region the most common symptoms are those of gastric disturbance. In the early stages these are usually indefinite in character and consist of discomfort after eating, vague pain, and nausea. As the tumor enlarges pain is more severe, and vomiting occurs.

In three of the cases there were crises of abdominal pain with vomiting much resembling cholelithiasis, but without tenderness over the gall-bladder, jaundice, or other symptoms of gallstones. The tumor in both cases was in the upper central region, displacing the stomach downward and backward.

In one case the sarcoma grew behind the lesser curvature and cardiac end of the stomach, and by its pressure diminished the capacity of that organ, so that the patient experienced a feeling of satisfaction and loss of appetite after taking a very small quantity of food. He had also dysphagia due to pressure upon the cardiac end of the œsophagus. The hydrochloric acid of the gastric contents was diminished, and this fact, combined with the presence of a mass, led to the diagnosis of gastric cancer and to the operation which revealed a retroperitoneal sarcoma growing behind the stomach.

When the tumor originates in the right or left upper quadrant pressure symptoms are not pronounced, and consist chiefly of digestive disturbances of the indefinite character already mentioned, nausea, vomiting, and vague abdominal pain.

The growths in the lower half of the abdomen give earlier signs of their presence and produce pressure symptoms which are more severe and more easily localized.

In retroperitoneal tumors of the lower right or left quadrants, the result of interference with the veins and nerves of the iliac fossæ are œdema and neuralgic pains in the foot and leg of the same side. Occasionally this pain is very much localized, especially in the early stages of the growth. In Rogowski's case the first symptom was pain in the right foot, most marked in the heel, and gradually spread-

ing over the whole leg. The tumor grew from the right iliac fossæ. Keresztszeghy's case, in the first series, was strikingly similar. The tumor was in the left iliac fossa, and the pain was at first very severe and localized in the left foot, and gradually spread over the leg.

In a case of the new series there was contraction of the adductor and flexure muscles in the leg on the same side, accompanied by severe pain. The tumor grew from the right iliac fossa.

Ellis reports the case of a boy, aged seven years, who had œdema of the left lung and of the left side of the face. The tumor surrounded the left kidney, but did not involve it.

In Osler's case there was polyuria without sugar, and the tumor grew in the median position. It seems probable that the symptoms in both of these cases were due to pressure upon the sympathetic system of the abdomen.

Femoral phlebitis occurred three times.

In Lunier's case blood was passed from the urethra, and on one occasion a piece of tissue was discharged through the same channel. Microscopic examination showed that this was from a lymph gland, and a diagnosis of tumor of a retroperitoneal lymph gland was made. Autopsy revealed a retroperitoneal sarcoma that had perforated the bladder.

Ascites may occur, but is not frequent. Hydronephrosis is also rare and occurred in but three cases.

Tumors of the pelvis and lower central abdomen produce early and definite pressure symptoms, as would be expected. Difficult micturition is one of the earliest and most common manifestations of retroperitoneal sarcoma in this region. It varies from frequent urination in the early stages to the complete obstruction of the neck of the bladder in the later stages.

Pelvic tumors usually produce constipation, which may amount to intestinal obstruction. Other cases, especially in the earlier stages, have persistent diarrhœa.

If the tumor encroaches upon both iliac veins there will be œdema of the genitalia and both legs.

Occasionally, if the tumor originates in such a position that it does not press upon any of the structures of the peritoneal cavity and retroperitoneal space, there are no pressure symptoms, properly

speaking, but merely evidences of disturbance with the function of the digestive tract. In such cases pain and œdema and the other signs of pressure appear later in the course of the condition, after the tumor has attained sufficient size to encroach upon the neighboring structures.

Physical Examination. In the earlier stages the physical signs of retroperitoneal growths are those of a deep-seated abdominal tumor concerning which little can be determined by palpation. Such tumors are rarely observed in their early stages, since their symptoms are so vague at the onset that medical advice is not sought until the tumor has grown to such a size that it is plainly perceptible to the patient himself.

The insidious onset of retroperitoneal sarcoma is a real source of danger, since by the time that the tumor is discovered it has grown to such a size that an operation is exceedingly difficult or impossible, and this is probably the reason why the results of operative treatment are decidedly unsatisfactory.

As the tumor enlarges its retroperitoneal position can usually be determined by the signs that are becoming definitely recognized as peculiar to growths originating in the retroperitoneal space; and the relation of the colon and other hollow viscera to the tumor is by far the most important part of the examination in determining whether or not the growth originated behind the peritoneum.

Since Witzel first called attention to the characteristic relation of the stomach and bowel to retroperitoneal tumors many writers have verified his statements.

The conclusion of my first paper as to the relations of the colon to retroperitoneal tumors proper agreed substantially with those of Witzel, except that several of the collected cases demonstrated conclusively that the transverse colon may be displaced forward by retroperitoneal tumors in the median position, although Witzel claimed that this could not be the case.

The most important systematic observations that have as yet been made upon this subject are those given in the recent classical paper by W. L. Harris, entitled "The Relation of the Colon to Intra-abdominal Tumors." Dr. Harris confirms the conclusions arrived at in the study of my first series, but has carried the subject farther,

and has included in his observations tumors that are not of retroperitoneal origin.

If the colon is carried forward upon the anterior surface of the tumor, it is proven almost beyond doubt that the tumor originates in the retroperitoneal space.

The stomach is displaced outward and to the left by upper central tumors, and coils of small intestines may be found upon the anterior surface of tumors that sprung from the root of the mesentery and grew up between the layers. However, the relation the stomach and small intestine bear to the retroperitoneal tumors is not as characteristic as the displacement of the colon to which reference has just been made.

The following is a classification of the relation of the colon to tumors originating in the various regions of the retroperitoneal space.

Upper central or median tumors sooner or later dissect up the peritoneum until they reach the transverse mesocolon, and then, as they enlarge and grow between the leaflets, the colon is carried forward upon the anterior surface of the same. The bowel usually runs across the anterior and lower surface and curves slightly downward, with the convex of the curve toward the umbilicus. But it may run along the anterior and upper surface, as in a case that has lately been under observation, where the transverse colon occupied the sulcus between the tumor and the liver.

If the tumor is not exactly in the median position, but grows a little to one side or the other of the median line, the transverse colon may cross the anterior surface of the growth diagonally.

Lateral tumors carry the ascending or descending colon forward upon their anterior and inner surface in the same manner. Then the colon curves slightly inward with the convex portion of the curve toward the umbilicus. Here, also, under some conditions depending upon the direction of the tumor's development, the colon may cross it diagonally.

The important fact is that the tumor has grown between the layers of the mesocolon and has carried the large bowel forward on its anterior surface. This cannot occur with tumors originating within the peritoneal cavity.

This relation of the colon to retroperitoneal growths is not characteristic of retroperitoneal tumors proper in the accepted sense of the term, as was insisted upon in my first paper. It is seen in tumors developing in the kidneys, in the adrenals, or in the accessory adrenals, in the remains of the Wolffian ducts, in pancreatic cysts, in abdominal aneurysm, and, in fact, in any growth originating in the retroperitoneal space, using the terms in their broadest sense.

Harris states that in 95 per cent. of thirty-four cases of pancreatic cysts the colon was carried forward and downward, but the relation of the colon to pancreatic cysts is not as characteristic as its relation to retroperitoneal growths developing farther down, since the pancreas lies almost entirely above the transverse colon, and consequently, the colon will not cross a pancreatic cyst as far up upon the surface of the tumor as it would in a case of retroperitoneal tumor originating in the middle of the retroperitoneal space.

A true retroperitoneal tumor may be so situated as not to affect the colon. In the growths originating in the upper central or upper left portion of the retroperitoneal space the stomach and not the colon is displaced. In such cases the stomach is pushed forward and to the left.

In tumors growing from the attachment of the mesentery and separating its layers, the small bowel surrounds the growth like a crown, and may even cover it completely, so that there is tympany in front of the mass, and as the tumor extends laterally and reaches the abdominal wall in the flank there will be dulness in this region, with the tympany in front, so that the whole picture suggests abdominal effusion. In several of such cases attempts have been made to draw fluid with the aspirator.

The presence of the colon between the tumor and the anterior abdominal wall can best be demonstrated by inflating the bowel with air through the rectum.

In the early stages the tumor is firm in its consistency, and is often nodulated or knotty, especially when it originates in the retroperitoneal lymph glands. The tumor may be very hard, but the term elastic is used with striking unanimity by the various observers in describing its physical characteristic.

In one case of my own the large and firm spindle-cell sarcoma originating in the upper central region possessed this peculiar elastic consistency, and resembled the liver so closely on palpation that the growth was supposed to be the liver enlarged from passive congestion.

In another case that I had the opportunity of observing the tumor was distinctly elastic without fluctuation except in one small area.

In the case reported by Jacoby a thrill was felt over the mass resembling that often observed in an echinococcus cyst. The autopsy showed a sarcoma with multiple area of degeneration.

Although in the earlier stages the tumor of retroperitoneal sarcoma is hard or elastic, there is a distinct tendency to degeneration, which occurs rather earlier in the course of retroperitoneal sarcoma than other solid tumors of the abdomen. This tendency to softening is distinctly characteristic of retroperitoneal sarcoma, and is practically the only point in the diagnosis by which it can be distinguished from other solid malignant tumors of the retroperitoneal space.

The degeneration is usually described as hemorrhagic, and unless the growth is removed in its first stage the autopsy reveals, in a majority of cases, a central cavity filled with a cheesy, brownish fluid containing old blood pigments or the evidences of recent hemorrhage.

In thirty of ninety-six cases the tumor was so degenerated that it had become practically a cyst, of which the walls were formed by the remains of the tumor mass, and the centre was filled with the fluid contents described above.

In twelve of the cases fluctuation of the mass as a whole was obtained before death, and in many more small softened areas were observed, although the rest of the tumor remained firm and hard.

As a rule, the tumor is immovable as would be supposed from its deep position and broad attachment. However, in fifteen of ninety-six cases the tumor moved on palpation, and in six it moved on deep inspiration.

The growth of the tumor is decidedly rapid. In fifty-eight cases in which the duration could be definitely determined the average length of time from the beginning of subjective symptoms to the

operation or death was 8.5 months. The shortest case reported by Chassaigne ran thirty-two days. McGraw reports a case of a man who had had a slowly developing tumor in the abdomen for five years, but in which the growth was very rapid in the last six months of life. It seems possible that the period of rapid growth in this case represented the true duration of malignancy. In the first case of my own we noticed no change in the tumor during the last fifteen months of life, although its development before this time had been rapid, and it occupied a space of about eight months. This case is also remarkable for the lack of cachexia, and for the very slight influence that the sarcoma had upon the patient's general health and strength. Indeed, her death was due to other causes than malignant tumor. It is probable that the patient's advanced age prevented the rapid tissue changes that would have occurred in a similar growth in a younger person.

Termination. The termination is usually death from exhaustion, unless there is surgical interference.

In three instances death resulted from the rupture of the softened tumor in the peritoneal cavity.

Tapping was resorted to in five cases. Aspiration offers some aid in differentiation between cysts and solid retroperitoneal tumors, but it is questionable whether the measure is justifiable in view of the fact that running a needle through the peritoneum into the tumor must be attended with some risk, and that the only hope of recovery in any malignant retroperitoneal tumor lies in operation.

In a certain number of cases wasting and anæmia appeared slowly and comparatively late in the course of the tumor's development, but, taken as a whole, cases of retroperitoneal sarcoma present the same degree of emaciation and cachexia that are characteristic of other malignant tumors.

Diagnosis. The differential diagnosis between solid malignant tumors of the retroperitoneal space and other tumors in the abdominal cavity which might be confused with them has been fully discussed in my previous paper, and a study of my own cases and of the additional cases collected from literature only emphasize the fact that a positive diagnosis as to the origin of solid malignant retroperitoneal tumors is practically impossible without exploratory incision.

Tumors of the kidneys and pancreas may manifest themselves by urinary or digestive disturbances peculiar to these organs. New-growths in the adrenals may give more or less definite symptoms of Addison's disease. However, tumors may develop in any of these three organs without characteristic clinical symptoms.

Retroperitoneal lipomata and the rare retroperitoneal congenital cysts may usually be distinguished by their physical characteristics, and tapping would be conclusive. Various rare solid non-malignant tumors of the retroperitoneal space have been reported, such as myofibromata and osteoenchondroma. The distinction between them and retroperitoneal sarcoma should be made by the history and course.

Tubercular enlargement of the retroperitoneal lymph gland should offer no difficulty in diagnosis. For a more detailed consideration of this subject see my previous paper.

Mesenteric cysts present a fluctuating tumor which lies at first laterally, but which enlarges and comes to occupy the middle of the abdomen, points toward the umbilicus, is freely movable, especially transversely, and rotates upon its axis. It is surrounded by a zone of tympany and has a zone of tympany passing over it. (Moynihan, *Annals of Surgery*, July, 1897.)

Solid tumors originating in the mesentery are very movable and are distinguishable by this characteristic from the retroperitoneal tumors originating at the base of the mesentery and growing up between its leaflets. In other respects, namely, the arrangement of the small bowel in front of the tumor, and the dulness in the flanks, simulating abdominal effusion, the two tumors are very similar.

Aneurysm of the abdominal aorta may very closely simulate a solid retroperitoneal growth. When the physical signs characteristic of aneurysms are well marked such a mistake could hardly be made; but when the aneurysmal sac has reached a great size, and has been so nearly filled with organized clots that the usual thrill, bruit, and pulsation are much lessened or entirely gone, such an error may readily occur.

Parker Syms reports a case of aneurysm of the aorta that so closely simulated a solid tumor that an exploratory operation was undertaken.

TREATMENT. The only hope for the patient lies in surgical interference and removal of the tumor. Naturally the earlier in the course of the affection that this is done the better the prognosis. Consequently the responsibility of making as early a diagnosis as possible lies with the medical man, and hence the importance of including retroperitoneal sarcoma in the list of possibilities when a case is under examination that shows indefinite abdominal symptoms with loss of weight. It is probable that physical examination in deep narcosis after thorough emptying the bowel might often reveal a retroperitoneal tumor at a much earlier stage than such tumors are generally discovered.

It is not my intention to enter into a discussion of the surgery of retroperitoneal sarcoma. It is sufficient to say that the only hope of recovery lies in the earliest possible diagnosis and removal of the tumor.

In the ninety-six cases here referred to operation was undertaken twenty-nine times, with eighteen deaths and eleven recoveries.

The survival of the patient for two months after operation has been arbitrarily considered a recovery.

CONCLUSIONS. *Frequency.* Sarcoma of the retroperitoneal space is not so very uncommon. In the past few years it has become recognized that the development of such tumors has a definite symptom complex, and more reference is being made to the condition in current medical literature, with a relatively larger number of reports of cases.

Etiology. The condition is more frequent in the first, fourth, fifth, and sixth decades. Fifty-three per cent. occurred in the interval from the thirtieth to the sixtieth year. Sex is not a predisposing cause. In ninety cases there were forty-four males and forty-six females. In four cases there seemed to be a direct relation between an abdominal injury and the development of the tumor. In one case the tumor grew from the wall of the retroperitoneal abscess.

Course. The development of the tumor is very quick. The average interval from the time the growth was first detected to operation or death in cases was eight and one-half months.

Position and Morbid Anatomy. The most common point of origin of these tumors is in the lumbar region, and the right side is

the oftenest affected. The next most frequent point of origin is the centre of the retroperitoneal space. Next comes the iliac region. In six cases the tumor grew in the pelvis, and in four cases it originated in the upper central region above the level of the umbilicus. The tumor was lateral in the majority of cases (fifty-six, or 60 per cent.), and two-thirds of the lateral tumors were upon the right side.

The tumor is almost always single. It is usually lobulated and capsulated, and is hard and firm in its earlier stages, but is very prone to degeneration. Degenerative processes are oftenest hemorrhagic in character, but may be puriform or myxomatous. In a third of the cases the softening progressed to such a degree that the growth assumed a cystic character.

Metastasis occurred in one-third of the cases. When it occurs the liver and lungs are oftenest affected. In many cases the growth involved the intestine, and the softened central area may rupture into the gastrointestinal tract or the peritoneal cavity.

Symptomatology and Diagnosis. The earliest symptoms are the presence of a hard or elastic nodular tumor usually quite immovable, growing deep in the abdominal cavity, and giving rise to rather indefinite digestive disturbances. If the mass is placed so as to interfere with the important structures of the abdomen or retroperitoneal space there may be characteristic pressure symptoms, such as pain and œdema in the legs, genitalia, or obstruction of the urethra or the large or small bowel. As the tumor enlarges the symptoms of interference with the abdominal viscera are aggravated, there may be constipation, sometimes amounting to intestinal obstruction, difficult micturition, violent vomiting, or signs of perforation of the bladder, stomach, or bowel.

The point of most value in the physical diagnosis is the relation of the colon and intestines to central or lateral growths and the relation of the stomach to upper central tumors. The determination of the position of the stomach or the colon in most cases should decide whether or not the tumor is retroperitoneal. When this has been done the diagnosis is narrowed to tumors of the kidney, adrenals, accessory adrenals, remnants of the Wolfian ducts, tumors of the pancreas, cysts of the pancreas, aneurysms and solid retroperitoneal tumors in the restricted use of the term.

Perhaps the only sign that is at all characteristic of retroperitoneal sarcoma, and that can be used in distinguishing between sarcomata and other solid tumors of the retroperitoneal space, is the tendency of the retroperitoneal sarcoma to degenerate early in its course, and when a tumor that is evidently retroperitoneal, and that was at first hard and nodular, rapidly softens and shows spots of fluctuation, the evidence is strongly in favor of its being a sarcoma springing from the retroperitoneal glands or connective tissues.

An exploratory incision is the only certain means of determining the origin of solid malignant tumors of the retroperitoneal space, and surgical interference offers the only hope of prolonging the patient's life.

The prognosis of the condition is distinctly unfavorable. The chief danger lies in the insidious development and lack of characteristic signs and symptoms in the early stages of the growth of the tumor. An early diagnosis is imperative if any benefit is to be derived from surgical interference. It is important, therefore, to recognize that such tumors are not excessively rare, and that their development presents a somewhat obscure but still characteristic symptom-complex. Hence, it is important to include retroperitoneal sarcoma in the list of possibilities in the examination of cases with indefinite disturbances of digestion and loss of weight, especially if there are signs of pressure upon any of the structures included in the retroperitoneal space.

DISCUSSION.

Dr. J. A. Scott: Dr. Steele has mentioned most of the causes of the origin of retroperitoneal sarcoma, and yet one has recently been under my care which took its origin in, I fancy, a rather unusual position. This man was aged about forty years, and had been in failing health for some three or four years. He had been considered more or less of an invalid during that time, though he had been hard at work. Some seven or eight months ago he had sharp, shooting pains in the right leg, and it was supposed that he had a sciatica, for which he was treated, and after a certain length of time the pain disappeared. He came under my care a short time ago, and only a few days before his death, with the evidence of what I thought was a con-

gestion at the base of his lungs, a rapidly failing heart, and subnormal temperature with great anæmia and apparently much prostrated. He died rather suddenly, apparently of an acute pulmonary suffocation. The lungs filled up suddenly, and I could not account for it, except from the standpoint of some acute infection, such as miliary tuberculosis, though the absence of temperature made me feel that there was no such condition. When the case came to autopsy we found the following condition of affairs: He had two undescended testicles. The right testicle entirely filled the pelvis; it was as large as an orange, and connected with the cord on that side. It had undergone apparently some form of sarcomatous degeneration that unquestionably was the cause of his sciatica, and accounted for pressure symptoms down his right leg. The tumor mainly occupied the right portion of the pelvis. The left testicle was undescended and normal. He had metastasis in the retroperitoneal space behind the stomach and above the hepatic vein, but not sufficiently large to produce pressure symptoms. There was no indication that he had had any œdema or ascites; but a gland had softened and ruptured in the inferior cava and flooded the lungs with sarcomata which varied from the size of a dollar down to a small pea.

The man had previously been under the care of very competent physicians, and I doubt if any of them had the faintest suspicion of the cause of his death, but that was the condition which was found; first, the origin of the sarcoma in the testicle; the second, growth in the retroperitoneal space; and, third, the sudden flooding in the inferior cava of this material which ended his life in a very short time from general pulmonary sarcomatosis.

PERFORATING GASTRIC ULCER; POSTERIOR GASTROENTEROSTOMY; FOWLER'S POSITION.[1]

By J. H. MUSSER, M.D., and W. W. KEEN, M.D.

MEDICAL HISTORY OF THE CASE AND REMARKS BY DR. MUSSER.

I have reported with Dr. Wharton a case that was in many respects similar to this. In that case operation was done within ten hours after perforation, which accident fortunately took place while the patient was fasting, an incident corresponding to the course of the second case. In the second case the operation was done five and one-half hours after perforation. The first patient had not had solid food for twenty hours, and only a small amount of coffee eight hours before perforation. The second patient had been without solid food, and had taken only albumin-water for from forty-eight to sixty hours. No opportunity to make a gastric analysis was given in the first instance, although it is presumed hyperchlorhydria was present, an extreme state of which was found in the second. Doubtless this excess of acid, unneutralized, contributed to the perforation. Accordingly, it may be well to emphasize the importance of neutralizing the acid by appropriate remedies in cases of gastric ulcer when food that takes up the acid is not permitted. Without doubt the early operations at the time when the stomachs were empty were important factors in saving the lives of the patients.

Another point of great interest was one of dissimilarity, but the contrast teaches an important lesson. The first patient was a

[1] Read February 3, 1904.

muscular farmer. Rigidity of the abdominal muscles was extreme. In the second case there was great atrophy of the abdominal walls, and in consequence there was no spasm or rigidity. Hence, given the lesions likely to cause spasm under other circumstances, it will be absent with abdominal walls of the character to be described.

I should like to add that the first patient is in excellent health, no gastric symptoms having returned.

I had seen Mrs. M. during November, at the request of her husband, a physician. She was seventy years of age, and had enjoyed excellent health until one year ago. She had been treated at that time by Dr. Boardman Reed for ulcer of the stomach, characterized by pain and hemorrhage. When seen by me she was suffering from pain, the occasional vomiting of large amounts of fluid, the daily regurgitation of a small amount. The pain was spasmodic, referred to the pylorus and relieved by regurgitation. It was not increased by food, except when the stomach was overfull.

On physical examination, the stomach, overdistended, could be well made out, and visible peristalsis was present. The pain often was coincident with the termination of a wave of peristalsis at the region of the pylorus.

I concluded that the patient had pyloric obstruction due to spasm. No tumor could be made out. A soft tube passed into the stomach resulted in the withdrawal of two quarts of dark, grumous, offensive material. Afterward there was perfect relief, and for almost three weeks she was without pain or distress. A test meal (Ewald) revealed total acidity 65, free HCl 40, absence of lactic acid, presence of sarcinæ and large bacilli. The odor was butyric. Lavage was done most carefully only twice, because of the possible ulcer. It seemed that an ulcer had healed, causing constriction of the pylorus, or that an active ulcer was attended by pyloric spasm. The small vomitings were due to the regurgitation—the incontinence of retention. November 17 the patient washed out her stomach herself without asking advice. November 19 sudden profuse hemorrhage occurred; at least a quart was vomited, followed by collapse. Rectal feedings, sips of water and egg albumen were resorted to.

November 26, at 9.30 A.M., she was seized with sudden pain in the medium line or to the right, between the xiphoid and the navel, followed by shock. I saw her within half an hour. There was pain, vomiting of mucus, but no blood, rapid pulse, tenderness, no spasm, cool extremities. She lay on the left side chiefly. The pain was considerable in the back and right shoulder. Liver dulness was present. A perforation had doubtless taken place, and an operation was advised.

Dr. Keen saw the patient at 12 M. Dr. Reed, her former attendant, joined us. Reaction had set in. Temperature, 99° F.; pulse, 85. The

patient complained of great pain and favored the left lateral dorsal position. Drs. Keen and Reed concurred in the diagnosis of perforation, and urged with me immediate operation. Dr. Keen operated at 3 P.M., five and a half hours after perforation had taken place.

SURGICAL HISTORY AND REMARKS BY DR. KEEN.

I first saw Mrs. M., a woman a little past seventy years of age, in consultation with Dr. J. H. Musser and Dr. Boardman Reed, November 26, 1903, at noon. I had known the patient for a number of years. I had not seen her for three or four years, and was struck with her loss of weight and emaciation. After an examination, I fully concurred in the diagnosis of perforated gastric ulcer, and three hours later the operation was done.

An incision was made to the left of the middle line on account of extensive diastasis of the recti muscles following her single pregnancy about forty-eight years ago. Only a very few muscular fibres were seen. The muscles were not only widely separated, but greatly atrophied. The entire abdominal wall was not over 1 cm. in thickness. As soon as the abdomen was opened a little turbid, rather dark-colored fluid poured out, and when the stomach was drawn out a few flakes of lymph were found on it. In a moment the perforation was discovered. It was an aperture slightly oval, very sharply punched out, 5 mm. in its long diameter, situated precisely in the middle of the pylorus anteriorly. The whole pylorus was thickened, but not nodulated. Evidently it had been the seat of an old ulcer involving all the anterior wall. The ulcer was immediately closed by a continuous Lembert suture. The abdominal cavity was then flushed out with a large quantity of salt solution, care being taken to wash out between the diaphragm and the liver, and also down into Douglas' cul-de-sac, where a considerable amount of turbid fluid was found. The diaphragm was but little injected. The transverse colon was then torn up, and a hole torn through the mesocolon, and a posterior gastroenterostomy done by means of a Murphy button. The peritoneal cavity was then again most carefully flushed out till the fluid, as in the first instance, returned clear. A number of adhesions were found between the coils of the intestine and the anterior abdominal wall and the omentum and the abdominal wall in the lower half of the abdomen, but I did not think it wise to prolong the operation by interfering with them. She was placed in bed in Fowler's position (*i. e.*, with head of the bed raised).

She made an uninterrupted recovery, her highest temperature being 101.4° F. On December 1, five days after the operation, her temperature reached normal. In consequence of the thinness of her abdominal wall I kept her in bed for four weeks. She went home December 31, wearing a belt and able to walk about without trouble.

The recovery from so serious a lesion as a perforating gastric ulcer and so serious an operation as abdominal section and gastroenterostomy in a woman past seventy is a sufficient reason, it would seem, for recording such a case. Her recovery was due, I have no doubt, largely, first, to the promptness with which Dr. Musser acted, and, secondly, to the fact that her stomach contained little beside albuminized water. Yet, in spite of this favorable condition of the stomach, there were a few flakes of lymph already present on the stomach. A culture was taken from the abdominal cavity as soon as it was opened, and this proved, after eight days, to be sterile. I do not know that Fowler's position influenced recovery in this case very much, in view of the facts just stated, but it certainly, at least, did no harm.

I debated for a few moments whether I should do Finney's gastroduodenostomy by means of a horseshoe-shaped incision extending from the duodenum into the stomach. I, however, finally decided against it on account of the great thickening of the whole anterior pylorus, and because I believed it would take longer than a gastroenterostomy by the Murphy button. For the last reason also I decided against a gastroenterostomy with simple suture.

Up to the present time, over two months after the operation, the button has not passed, but it has not produced any trouble.

DISCUSSION.

Dr. John H. Gibbon: Until a year ago I had had no personal experience with perforated gastric ulcer, but during the past twelve months I have operated upon three cases of perforated gastric ulcer and one of perforated duodenal ulcer. There are two points which have greatly impressed me in the method of treating this catastrophe. One is the great necessity of early operation; and the second is the resemblance which a gastric ulcer accompanied by marked induration bears to malignant disease. Delay in operating may be due to the difficulty of diagnosing the condition, and yet in most cases, although we may not be able to determine the exact lesion, the indications for operation are plain enough. In two of my cases there was absolutely no history pointing to gastric ulcer, and this is frequently the case. In two of the cases a very positive history was given, and in a third a diagnosis was also made even in the absence of history.

In a fourth case, the duodenal one, a diagnosis of appendicitis was made. The literature on the subject shows this to be a very frequent error, the majority of cases of duodenal perforation being mistaken for appendicitis. In all of the cases the indications for operation were clear. Two of the four cases recovered, and a third would have recovered except for faulty technique. This patient died of a pelvic abscess three weeks after the operation. The pelvis was drained in each of the cases, but in this instance it was not sufficiently thorough. The other fatal case was not operated upon until thirty-three hours after perforation, and was then in a bad condition from general peritonitis. Death occurred in this instance eleven hours after operation. I believe this patient, had he been operated upon as promptly as the others, would have made a good recovery. This group of cases I have reported in *American Medicine* for December 19, 1903. In addition to pelvic drainage in all of these cases I employed the Fowler position, that is, the head of the bed well elevated. The second point I call attention to was well illustrated in the last case operated upon. Here the induration about the perforation was so great and so hard that had I opened the abdomen in the absence of perforation I would certainly have considered it a case of carcinoma of the gastric wall. Such errors in diagnosis have frequently occurred and account for the remarkable recoveries after operation for supposed malignant disease where no attempt has been made to remove it. I performed a gastroenterostomy upon a patient last summer in which I was sure that I was dealing with a malignant disease of the pylorus well advanced. The mass was as large as a lemon, and there were enlarged glands in the lesser omentum. This woman made a very prompt and satisfactory operative recovery, and for a number of months has suffered from no gastric symptoms, although she is taking ordinary diet. She has put on flesh and is now considering a return to her ordinary work. Continued improvement in this case causes me to feel that the mass I encountered at the pylorus was not malignant, but the result of induration about a pyloric ulcer. The history in this case was typically that of a pyloric ulcer.

Dr. Stewart: I have operated upon three cases of gastric perforation during the past year, with recovery in each instance. In all of the cases the pain was referred to the right iliac fossa, and in two cases a diagnosis of appendicitis had been made before I saw the patients. In all these cases the operation was very prompt (within five hours), and in each case the patient had a stomach full of ordinary food. The first patient was a boy, aged eleven years, who was shot in the left hypochondrium just after eating a large dinner. The bullet perforated both walls of the stomach. The symptoms were rigidity of the abdomen, tenderness in the epigastrium, pain in the back and in the right iliac fossa. He vomited a large part of his dinner, but no blood. Both perforations, one in the anterior and one in the posterior wall, were closed by purse-string sutures and the abdomen

was sutured without drainage. The purse-string suture is a ready method for closing perforations of this sort, but is often inadequate when the perforation is due to ulcer, the suture tearing out.

The second patient was a young man, aged twenty-four, who just before dinner had some pain in the abdomen. After dinner there ensued violent pain in the epigastrium, reaching to the back, rectum, right shoulder, and into the right iliac fossa. He was seen by an eminent surgeon who made a diagnosis of appendicitis, and who sent him to the hospital, where I saw him. He had had severe indigestion for about five years, although he had never vomited blood. When I saw him there was absence of liver dulness, dulness in the flanks, rigidity, tenderness, and collapse. This man had also vomited part of his dinner, but no blood. There was turbid fluid in the abdomen, but no odor or gas. The perforation was on the anterior wall near the pylorus, and it was closed with some difficulty. Two drains were inserted, one in the epigastric region and one in the pelvis.

The third case was a man, aged thirty-two years, who never had symptoms of indigestion. He was a bartender by occupation and denied the use of alcohol. After eating peas and chops he was suddenly seized with pain in the epigastrium, shooting down into the right iliac fossa. He vomited his lunch, but no blood. There was tenderness and rigidity; the leukocytes were 19,000; blood pressure 235; liver dulness present; no dulness in flank. The perforation was in the pyloric region in the anterior wall. This was sutured and drainage instituted.

Dr. William J. Taylor: In one case in which the ulcer was very near the pylorus and the whole pylorus and duodenum were greatly thickened, I was quite convinced that the mass was malignant. The patient was seized suddenly, about three o'clock in the morning, with intense pain, and was taken at once to the hospital. I did not see him until the afternoon. He had intense abdominal pain and rigidity, with tenderness in the right iliac fossa. I believed it to be a case of perforated appendicitis. The appendix was much inflamed and in such condition that demanded its removal. It did not seem to me, however, that all his symptoms could be accounted for by the appendix. Therefore, I searched farther up for the gall-bladder, thinking it might be perforated. Further search revealed an ulcer about half an inch in diameter, as though it were punched out in the wall of the duodenum, and from the general appearances I was convinced that the condition was malignant. The man's condition was such that I could only close the ulcer, and I did not do a gastroenterostomy as I should have done. Strange to say, the man made a complete and perfect recovery, and is well at this time.

The operation was done during the first week of November. The history he gave, and which I only found out afterward, was of long-standing trouble in his stomach and indigestion. He is now perfectly well and gaining flesh, and is better than he has been for years.

Dr. Richard H. Harte: I have listened with a great deal of interest to this report, and to my mind it brings out one point which ought to be emphasized: That dyspepsia is not necessarily a medical disease. I feel convinced that in a great many of these cases if they were turned over to the surgeon they would be more readily relieved, and that there is always some definite cause for this persistent indigestion, either appendicitis or gastric ulcer. A great many of these gastric ulcers, if operated upon, would not go on to perforation. If gastroenterostomy were done they would become perfectly well and all symptoms would disappear. In conversation with Mr. Moynihan, who has had an enormous experience in this work, he gave a history in all cases of chronic indigestion, and cited a case in which the patient had been in the hands of London specialists for all sorts of gastric treatment, but who after having gastroenterostomy done was relieved of all symptoms and immediately began to rapidly increase in weight.

Dr. Musser: I have nothing further to say. No doubt, as Dr. Harte pointed out, many cases of indigestion, particularly those characterized by hyperacidity, are due to ulcer, but not as many in this country as in England, for etiological reasons and because they get better under medical treatment.

I was glad to have the statement made by one of the speakers regarding the non-malignancy of many of the masses which appear to the naked eye to be malignant. Dr. Mayo himself has told me he is satisfied that many of the inflammatory conditions of the stomach are associated with enlarged glands, and hence their presence does not imply that the nodule in the wall of the stomach is of malignant origin.

I want to say a word about the great number of cases of gastric ulcer that have come to me this year, and a few words as to whether the surgeon can hold out any hope of relief in those cases in which the ulcer is situated back on the pancreas. In two of my cases there was death from hemorrhage which took place directly in the pancreatic duodenal artery. An extraordinarily large vessel was seen in the floor of the ulcer. In one case Dr. Flexner made the autopsy. Another case occurred in the Presbyterian Hospital. From the dissections which Dr. Flexner made it was thought that surgical relief could hardly have been given under such circumstances.

A CASE OF STATUS LYMPHATICUS.[1]

By J. H. MUSSER, M.D., and J. T. ULLOM, M.D.

T. D., aged five years and eleven months, when about eighteen months old had a protracted enterocolitis which ran a prolonged febrile course; had also a discharging ear as a resultant of an attack of influenza, and had had occasional slight febrile attacks attributed to digestive errors. After his enterocolitis he was very anæmic and emaciated, but never had rickets. Took on flesh rapidly and grew rapidly, and at the time of his fatal illness was very well nourished and large for his age.

Family History. Singularly prone to cardiovascular disease both on the maternal and paternal side.

Present Illness. On the evening of November 10 he became hoarse, and the hoarseness continued throughout the night, but on November 11 he seemed much better and played as usual all day. He was seen about 6.30 or 7 P.M. of that day by the family physician, and his temperature was 100° and his pulse 100. The pulse seemed a little rapid for the temperature, but it was usually out of proportion to any fever, and little attention was paid to it. He went to bed as usual and slept soundly, but at 2 A.M. his mother noted that he looked pinched and that there was a great deal of rattling and bubbling apparently in his throat. No alarm was felt, however, until about 4.30, when, though still sleeping, it was noted that he was very much cyanosed in his face and hands, and that his breathing was rapid and short.

The physician saw him about 5.30 A.M., and at that time he was in a state of shock and had some difficulty in breathing, respirations 45 to 50, pulse 160, quick and weak, some cyanosis of the face and hands. The bubbling and rattling in the throat and chest were very evident. He was asleep, but when aroused his mind was perfectly clear. His voice sounded hoarse. At 7 o'clock he was much the same, his temperature being 102°, pulse 150, respirations 50. He was extremely restless, tossing about in his bed. His voice was at times hoarse, and again would become perfectly clear. At 9 o'clock he was about the same. He was actively stimulated

[1] Read March 2, 1904.

with strychnine, whiskey, and atropine since the last observation. His pulse was 150 to 160, respiration about 60. His voice was clear and hoarse at intervals, as before noted.

Physical Examination. Boy, very large, apparently older than five years. Clear complexion, the skin being very clear and the veins very distinct. He was very sturdily built, being what is ordinarily called a "fat boy." He was very dyspnœic, and his face and hands were somewhat cyanosed, and pupils dilated. Waxy pallor of the hands and face was most apparent. There were bubbling and rattling in the throat and chest, such as is heard in ether anæsthesia. There was retraction of the interspaces and epigastrium on inspiration, with labored inspiration and expiration. It required great effort to breathe. He was very restless, and even during the physical examination tossed all over the bed. There was considerable movement of the larynx. His pharynx and larynx showed no membrane, but were congested.

Palpation. Practically negative.

Percussion. Revealed no areas of dulness, indeed a hyperresonant note all over the chest, although the observations were made with difficulty.

Auscultation. Only a confused bubbling and rattling could be heard, no bronchial breathing. There seemed to be apparently mediastinal pressure, from the labored breathing.

A diagnosis of probable hypertrophy of the thymus or of status lymphaticus in the course of some infection was made, and, as the possibility of a laryngeal tracheal diphtheria was thought of, he was given 3000 units of antitoxin, and was heroically stimulated. His pulse now was 160 and very weak, and respirations 60 to 70. After the stimulants there would be some response, but after a few minutes the pulse would be as weak as before. His condition grew progressively worse, and at 12.30 he had a slight convulsive seizure. He seemed now to be entirely oblivious to his surroundings, although until this time he had spoken to those about him and recognized them. These slight convulsive seizures continued at short intervals (five or ten minutes). He was placed in a mustard bath, and responded to this very well, his pulse becoming stronger and less rapid, about 140, and his convulsions ceasing temporarily. About 1 P.M. his temperature, taken in the axilla, was 105.6°. His stimulation had been carried on regularly throughout all this time, but he went rapidly from bad to worse, and died peacefully at 2.30 P.M. His urine, obtained by a catheter at 1 P.M. and examined after death, was loaded with albumin, but showed no casts. Autopsy performed at 5 P.M.

Autopsy. (Made by Dr. W. T. Longcope). The body is that of a young lad, about 120 centimetres in length. Rigor mortis present. Moderate post-mortem discoloration over dependent parts. Pupils equal and dilated. No œdema of extremities. Subcutaneous fat present in good amount, of orange-yellow color. Muscles dark.

Abdominal Cavity. Omentum contains much fat, and entirely covers the intestines. Peritoneal surfaces are everywhere pale, smooth, and glistening. The small intestines are slightly distended with gas. Appendix lies over the brim of the pelvis, is patent throughout, and appears normal. Diaphragm on right side reaches the third rib, on left side to third interspace.

Thorax. On removing sternum, lungs collapse slightly. The left pleural cavity is free from fluid and adhesions. A few cubic centimetres of turbid fluid of a yellowish color are found in the right cavity and over the posterior portion of the lung. The parietal and visceral pleura are adherent by a few delicate fibrinous adhesions. The superior mediastinum is almost entirely filled by the thymus gland. The general appearance is of three lobes: a middle lobe which lies, as far as can be seen, just below the sternal ends of the clavicles and reaches almost from one clavicle to the other, being about 1 centimetre nearer the left than the right. What appears as the lateral lobes extend down into the mediastinal fat, between the pleura and pericardium. The right lobe measures 6 centimetres in length and extends to the upper margin of the third costal cartilage. These lateral lobes are more or less fused above the pericardium, but as they descend they separate and apparently extend far down between the pericardial sac and lungs. Lower down the phrenic nerve is in intimate connection with the gland on both sides.

The pericardial cavity contains a slight excess of fluid, clear and straw-colored. The serous surfaces are smooth and glistening.

Heart. The heart is of moderate size. Epicardium contains a slight excess of fat. Over the tip of the left ventricle is a soft, raised, milk patch about the size of a 5-cent piece. The heart contains firm, red, post-mortem clots and a small amount of fluid blood. All the valves are thin, delicate, and normal. The heart's flesh is firm, brownish-red, and rather dark in color. The aorta is smooth.

Auricular appendages free from thrombi.

Lungs. The left lung is of medium size, pleura is smooth and glistening, free from adhesions, and most of the upper lobe and anterior portion of lower lobe is pale pinkish-gray, rather puffed up and crepitant. In the posterior portion of the lung, and most extensive in the lower lobe, there are many irregular firm areas which are of a deep purplish-blue color, and sharply defined from the gray crepitant lung. The smaller ones measure 0.5 to 1 centimetre in diameter, and are slightly depressed. The larger ones average from 4 to 5 centimetres in diameter, are raised, and have a slightly lobulated appearance. On section the general cut surface is deep pink, moist, somewhat irregular, but crepitant. Here and there in the anterior portion of the lung small, deep purplish-black nodules are seen, which are raised, well defined, and measure from 3 to 5 millimetres in diameter. When they occur near the surface they correspond to the

depressed bluish areas. In the posterior part of the lobes there is a very irregular and extensive patchy consolidation of a purplish-black color. These consolidated areas are raised and have a smooth, slippery surface, which is irregular and appears somewhat lobulated. Scattered everywhere through the consolidations are seen small gray or yellowish points, the largest measuring from 2 to 3 millimetres in diameter; some of them exude drops of yellow pus. The smaller bronchi in these areas frequently contain pus, which can be squeezed out in thick, yellow drops. The bronchi at the root are deeply congested and contain a thick, yellow, mucopurulent material, which is most plentiful in the smaller ramifications. The vessels at the root are clear. The bronchial glands are not specially enlarged; they measure from 3 to 8 millimetres in diameter and are pinkish-gray in color. The right lung is of medium size. The middle lobe and most of the anterior portion of the upper lobe is pale pinkish-gray, dotted with a few small, deep purple areas, but is generally insufflated and crepitant.

On the under surface of the middle lobe along the extreme margin there are several large air bullæ. The lower lobe is very heavy, entirely solid, and of a deep purplish color. It has a rather nodular feel. In the posterior portion of the lobe there is a rounded, particularly raised area about 5 centimetres in diameter. This has a mottled, reddish-black, gray, and yellow appearance, while the pleura over it is roughened by a delicate film of fibrin. For some distance about this area the pleura is filled by a fine granular exudate, but elsewhere is smooth and glistening. On section the upper lobe and middle lobe show irregular areas of deep purple consolidations studded with minute yellow points, and in general the cut surface appears much like the section through the left lung. The cut surface of the lower lobe is deep purplish-red in color, but has a somewhat nodular look and feel, due to small raised masses from 3 to 8 millimetres in diameter, which are of a yellowish-gray color.

The general consolidated surface is smooth and slippery, but the raised nodules have a more or less granular look and seem to be made up of many small, discrete, and confluent granules. Beneath the area over which the pleura shows a fibrinous exudate the section exposes a large raised granular patch 2.5 centimetres in diameter, which is thickly sprinkled with minute yellow abscesses. The smaller bronchi, in many places, are entirely plugged by thick, yellowish pus. The bronchi at the root are deeply congested and contain a small amount of the same material. The vessels are clear. The lymph glands at the root are somewhat larger than those on the left side, but have the same general appearance.

The tonsils are apparently normal. The epiglottis is œdematous and deeply congested. It is covered by a good deal of mucus and a soft, yellowish, purulent material which is readily wiped off. The larynx is also deeply congested, being dark purplish-red in color and very œdematous, so

that the ventricles are almost closed, but on pressure a small amount of their purulent fluid wells up over the swollen vocal cords.

Small flakes of mucopurulent material cover the mucous membrane of the larynx, but there is no ulceration and no false membrane. The trachea shows the same condition as the larynx and the epiglottis, and this extends down into the bronchi. The right lobe of the thyroid gland and isthmus appear normal. The left lobe of the thyroid gland measures about 4 centimetres in length. Attached to its lower border is a smooth, rounded mass which measures about 2 centimetres in diameter, and appears about the size of a Lima bean. On careful dissection this does not appear to be in direct connection with the thyroid gland.

On section the cut surface is rather firm and has a slightly yellowish-gray color, being somewhat translucent. It is dotted with minute points, opaque and of yellowish hue.

The thymus gland appears very large, and is more or less divided into three lobes. The upper and middle lobe arises 1.5 centimetres below the lower edge of the thyroid gland, and measures 5.5 centimetres in length and 2 5 centimetres in width. The left lower lateral lobe measures 9 centimetres in length and 4 centimetres in width. The right lateral lobe measures 7 centimetres in length and 4 centimetres in width at its widest point. These two lateral lobes extend down in a wing-like manner from the upper and central lobe.

The thymus weighs 40 grams. On section it is of a pale pinkish-gray color, dotted with minute opaque yellow points. The consistency is rather firm.

Spleen. Weight 50 grams. Size, 8 x 5 x 3.5 centimetres. The spleen is of medium size, decreased in consistency, and of a pale lilac color. The capsule is smooth and free from adhesions. On section the pulp is soft, of a dirty-red color, and pliable rather than friable. Here and there small hemorrhagic points are seen. Malpighian bodies are large and well defined. The trabeculæ are not increased.

Liver. The liver is of normal size, regular in outline, decreased in consistency, and dark brown in color. The capsule is smooth and free from adhesions. On section the cut surface is smooth, dark brownish-red in color, and oozes much blood. The lobules are fairly well marked and have deep-red centres.

Portal connective tissue not increased.

Gall-bladder is distended with dark-green bile. Bile-ducts patent.

Kidneys. Both alike; combined weight 100 grams. The right kidney measures 8 x 4½ x 3½ centimetres. It is of medium size and decreased in consistency. The capsule strips readily, leaving a smooth, dark reddish-gray surface, covered everywhere with injected stellate veins. On section the medullary pyramids are pale about the papillæ, but deeply injected about the peripheral zone. The cortex averages from 5 to 7 millimetres in thickness, is grayish-red in color, and cloudy; striæ are fairly well marked; glomerule injected; pelvis and ureter are apparently normal.

Pancreas, stomach, and œsophagus apparently normal. Aorta smooth. Intestines apparently normal.

Anatomical Diagnosis. Acute mucopurulent laryngitis, tracheitis, and bronchitis; acute diffuse bronchopneumonia; hypertrophy of thymus gland; congestion of liver and kidneys; acute splenic tumor, and cloudy swelling of viscera.

Histology. Heart muscle shows œdema, fragmentation, absence of cross-striation of fibres and marked granulation of muscle cells.

Lungs show extensive bronchopneumonia of hemorrhagic type. Collections of staphylococci are scattered through the consolidated parts, and above them the lung tissue and exudate in air cells are necrotic.

The vessels show no especial changes. Bronchi show marked alterations, consisting in thickening of mucous membrane with infiltration of polymorphonuclear leukocytes, small round cells and epithelioid cells, while the superficial portions are necrotic, and epithelium is partially or wholly desquamated. There are many cocci in groups, found in sections stained by Weigert-Gram, which are scattered through the narcotic mucous membrane and seen in the lumen.

The trachea and larynx show practically the same condition as the bronchi. Mucosa is partially necrotic and covered with masses of cocci in groups. There is practically no formation of fibrin and no pseudomembrane. No bacilli can be found. Small lymph glands surrounding the trachea are infiltrated with polymorphonuclear leukocytes, and one gland is converted into an abscess. The bronchial and cervical lymph glands show hyperplasia of the lymphoid follicles with swelling and necrosis of the germinal centres. There is endothelial proliferation, and the lymph sinuses are large and packed with polymorphonuclear leukocytes, epithelioid cells, red blood cells, fibrin, and coagulated serum.

The thymus gland shows very little change from the normal, microscopically. The centres of the lobules are perhaps slightly enlarged, and the lymphadenoid tissue about the periphery hyperplastic. The spleen is congested, and the pulp is rich in lymphoid cells and epithelioid cells. The Malpighian bodies are swollen and the germinal centres are extremely large. There is extensive necrosis of the cells of the germinal centres.

Liver shows congestion accompanied with cloudy swelling and fatty degeneration.

The kidney shows marked cloudy swelling and congestion. There are no proliferative changes.

Microscopic Diagnosis. Extensive pneumonia with areas of necrosis. Acute inflammation and superficial necrosis of the mucous membrane of the larynx, trachea, and bronchi, acute cervical and bronchial adenitis, simple hyperplasia of thymus gland, congestion of spleen, with central necrosis of Malpighian bodies, parenchymatous degeneration of liver and kidney.

Bacteriological Examination. From lung and mucous membrane of larynx, staphylococcus pyogenes aureus. No other organism. Cover-slips from larynx and lungs showed only cocci in groups. No other bacteria. No growth of bacteria was obtained from heart, spleen, liver kidney, or thymus gland.

In taking up for consideration this very interesting condition, our attention turns to its etiology, pathology, diagnosis, and cause of death.

The condition has received such a limited amount of study, and the cases reported are so few, that little regarding its etiology can be gained from an analysis of the cases, and theories as to its cause must, at best, be vague and uncertain. It is considered a disease of childhood, and probably it is usually found in early life; but that it does occur in adults is without any doubt, for of eighteen cases available we found six occurring in patients between twenty-seven and fifty-five years of age.

Rickets is supposed to be a predisposing factor, or at least to be usually coincident, but in these cases we failed to find one with a history of rickets. A careful analysis of eighteen cases shows twelve males and six females, and the ages vary from eighteen days to fifty-five years. There was negative family and personal history in thirteen, and of the others, one, a male of three-and-a-half months, had a history of alcoholism in the mother, and both grandfathers died of phthisis. One, a male of forty, had a personal history of alcohol. One, a male of thirty, had had syphilis, rheumatism, and smallpox, and used alcohol to excess. One, a lad of thirteen, had a history of dyspnœa on exertion; and one, a boy of five, had a history of Bright's disease and organic heart disease, and tuberculosis on the father's side, and Bright's disease on the maternal side, and a personal history of enterocolitis.

Of the eighteen cases, nine died suddenly without apparent cause; five of anæsthesia; one of complicating tetanus; one after an illness of several months in which dyspnœa and aphonia were the principal symptoms and for which tracheotomy had been done, and in whom at autopsy an aneurysm of the thoracic aorta was found which exerted no pressure; one died of a condition diagnosed as laryngismus stridulus of a few hours' duration, and this probably

belongs in the class of cases dying suddenly without apparent cause; one case, the one reported, died with complicating bronchopneumonia.

In the nine cases of sudden death, six were children and were found dead in bed after having gone to sleep in apparent health; one was a male thirty years of age, who became unconscious while having his boots blacked and died on the way to the hospital; one was a lad, aged thirteen years, who while in apparently the best of health had a few slight convulsive seizures and died; one was a woman, aged thirty years, who died suddenly twenty-two hours after a normal labor. There were five cases from anæsthesia, the anæsthetic not known, the operations being thyroidectomy, circumcision, repair of vicious non-union of the humerus, supravaginal hysterectomy, and operation for necrosis of jawbone. The death in each case seemed due to sudden cardiac and respiratory failure.

Kundrat reports ten deaths from chloroform in which the thymus was enlarged.

Pathology. The pathological findings in these cases were practically constant, and consist of an enlarged thymus, spleen, lymph glands, Peyer's patches, tonsils, and pharyngeal lymph tissue. Not all these are found in every case, the enlarged thymus, spleen, and some of the lymph glands being constantly found. Cloudy swelling of liver and kidney also is a constant lesion. Histologically we find hyperplasia and proliferation of the lymphadenoid tissue of the thymus, lymph gland, and spleen, with focal necrosis of the germinal centres of these organs.

The question arises, Does this case conform to the pathology of the disease? We think it does. The one thing lacking is the focal necrosis and marked hyperplasia of the germinal centres in the thymus. But we think this can be overlooked when there is marked enlargement of the thymus and the changes in spleen and cervical and bronchial glands are identical with those found in other cases. The pneumonic process, the changes in the heart muscle, and the condition of the air-passages we will take up under the causation of death.

Diagnosis. The diagnosis of the disease can be made, but an opportunity is rarely given. Dyspnœa, dulness over the manubrium,

and enlargement of the superficial lymph glands are the factors on which we may base our diagnosis. Suddenness of onset, a transparent skin with prominent veins, and the absence of other explanations of the symptoms, are minor factors in the conclusions.

Ewing has claimed a lymphocytosis in the disease, and offers in support a case in which the lymphocytes, small and medium, were 84 per cent. of the leukocytes present, but the evidence of one case is hardly convincing. Hoarseness was noted in our case, which was transient, and in another case, a man aged thirty years, there was complete aphonia. In our case the hoarseness was due to the laryngitis, and its cause was probably to be found in the infection and not in the disease under consideration.

Cause of Death. The cause of death is probably the most interesting point in this rare condition. There has been a great deal of debate on the matter, one set of observers assigning pressure by the thymus, another toxæmia, as a cause of death.

Schule claims that it requires 1000 grams to compress the trachea, and there is no case on record where the weight of the thymus was above 150 grams. In twelve cases where the weight was given, the average weight was 54 grams, the highest 150 grams.

The thymus normally, according to Biedert, weighs at birth 14 grams, at nine months 20 grams, and at two years 27 grams. After the second year it remains about constant till puberty, after which it atrophies.

It seems indisputable that the thymus does cause pressure on the trachea, as witness the case of Koenig and Purrucker, in which operation relieved the dyspnœa. Lange reports a case where the trachea was compressed from left to right and from behind forward until one diameter was only one-third of the other. That it may cause dyspnœa by pressure on the phrenics or pneumogastric may be possible, but has never been proven. The cases of Koenig and Purrucker were both in young infants, one three weeks and one three-and-a-half months old.

The other set of observers claim death to result from toxæmia induced by the thymus or lymphatic apparatus. This opinion is not without very plausible evidence in its favor. Lochte, in some experiments on hyperthymization of the blood, found that this

caused, first, a lowering of the blood pressure due to paralysis or depression of the vasoconstrictors; secondly, acceleration of pulse due to direct influence on the heart; thirdly, an overdose killed, showing results at autopsy suggestive of asphyxia.

Flexner in some experiments with mydatoxins and cytotoxins found that their introduction caused a hyperplasia of the lymphoid tissue of the lymph glands and spleen, with focal necrosis of their germinal centres.

Blumer has called attention to the similarity in the lesions found to those of status lymphaticus. There is of course no change noted in the thymus, but the changes in the spleen and lymph gland are practically identical.

It is hardly conceivable that in those cases dying very suddenly, without any previous evidence of disease, the thymus could swell to sufficient bulk within a very short time to compress the trachea or cause dangerous pressure on the phrenics or pneumogastric, and none of the autopsies would further this view. On the other hand, that there is more or less of an autointoxication present, and that a sudden accession of toxic material is sufficient to disturb the physiological equilibrium and cause sudden death, is not an unwarrantable proposition.

It might well be asked what gives rise to this sudden accession of toxic material, and in most of the cases we must confess we are unable to answer. Whether there is a sudden cumulative effect or not, with some unknown factor as a cause, we find it impossible to say.

In our case there was no evidence of compression of the trachea, while there was a sudden onset and a coincident bronchopneumonia. There was a cardiac failure from the beginning, and there can be little question that this was the main factor in the cause of death. The heart muscle was degenerated, and we naturally seek the cause of this condition. There was an infection of the larynx, trachea, and pulmonary tissue, with consolidation of one lobe and small patches throughout the lung. Our alternative is a toxæmia, and we have two possible sources for that—one the infection, and the other the toxin, the product of the lymphatic system. While the source of the toxæmia might have been the infection, yet the experiments of

Lochte in hyperthymization proved that there was a direct depressant and paralyzant effect on the heart muscle, and until there is a further study on the subject no one can deny the plausibility of a theory of an autointoxication with its source resident in the lymphatic apparatus. In fact all our clinical experiences show that an infection of the type we had to deal with would not have caused such toxæmia in a few hours.

The question arises as to whether the condition of status lymphaticus causes a lowered vital resistance, thus rendering the organism more subject to an infection, or whether the infection was a mere coincidence and became a factor in causing death.

Two positions are open to us. We can say that the infection destroyed the vital equilibrium and permitted the toxæmia due to the thymus to kill, or we can say that the lowered resistance due to the thymus permitted the infection to kill more quickly than it otherwise would. Longcope has found that in chronic Bright's disease and cirrhosis of the liver there is a decrease in the serum-complement or alexin in the blood, and this fact permits of an easy infection, in most cases a terminal one. It is not hard to trace a similarity between such diseases and this one of status lymphaticus. The question is one, however, that requires a more complete study and observation of a large number of cases. The condition in the larynx unquestionably gave rise to the hoarseness, and the laryngitis was only a part of the staphylococcus infection.

REMARKS BY DR. MUSSER.

The practical point was this: A child apparently robust, with signs of laryngitis and bronchitis, but without high temperature, is after an illness of a few hours evidently dying, rapidly overwhelmed by some cause not readily demonstrable. Death was evidently imminent because of some toxic agent spending its force upon the circulation—a cardiac death. We could not realize it was due to an infection of the pneumococcus or streptococcus type, because of the clinical course, the low temperature, and the physical signs. That bronchial diphtheria was present was more than likely because of this low temperature, the rapid course, and the profound toxæmia, although we believe, if it was diphtheria, an albuminuria should have

been present with the first examination of the urine. Moreover, no etiological facts supported this assumption. Some other agent than that of an infection had to be involved, and so we concluded, because of the lymphatic type of the patient, because of the rapid onset of shock in the course of a mild infection, because of the seemingly manifest signs of mediastinal pressure and the absence of uræmia, the case was one of lymphatic constitution with enlargement of the thymus gland, in which status lymphaticus had taken place. In the light of the autopsy, although we did not realize such great infection of the lymphatic system, we were supported in the contention. We must confess, from the character of the respiration, we thought there was occlusion of the bronchi from intrathoracic pressure. Because of the age, the physical appearance of the patient, the sudden onset, and the absence of pleural effusion, we thought the thymus gland was enlarged and pressed upon the bronchi. Perhaps, had we remembered the aphonia would come and go, we might have excluded pressure. On percussion the high degree of secondary emphysema and circular shape of the chest permitted only a highly tympanitic note to be heard.

When the autopsy disclosed a thymus gland fully one-third larger than normal, there was no gross indication of pressure upon the other structures. It is possible with the agonal emptying of vessels any signs of marked pressure might have disappeared. Even though obstruction of the air-passages from within or without was present, death was overtaking the patient because of shock and cardiovascular failure, we believe, due to the lymphatic toxæmia. The diagnosis during life was that of status lymphaticus or lymphatism with enlarged thymus gland, in the course of which an infection causing laryngitis, tracheitis, and bronchitis occurred.

DISCUSSION.

Dr. S. Solis Cohen: This rare condition, overwhelming with death the patient, whether adult or infant, calls for as much exact study as it can receive. All such papers as this are therefore to be welcomed as important contributions.

The one thought that occurs to all is, What can be done to avert death

I could not follow entirely the discussion of therapeutic measures in Dr. Ullom's communication, and hence do not know whether the question of surgery arose in the case reported, nor am I at all sure in the light of the necropsy, with the complications described, that surgery would have been of the slightest value in that case, though it has been in others in which the thymus gland was much enlarged.

The question of thymic intoxication and its possible antagonism by some other internal secretion or by a thymolysin or antitoxic serum prepared by experimental immunization of animals deserves extended laboratory study. Thymus depresses the heart and dilates the vessels; adrenal stimulates the heart and contracts the vessels. It is thus a symptomatic antagonist. In exophthalmic goitre the thymus sometimes enlarges, as it does in hypertrophic pulmonary osteoarthropathy and in acromegaly—perhaps as a compensatory effort to maintain physiological equilibrium. Thymus also helps to control the symptoms of exophthalmic goitre, but so does adrenal; while thymus may be used instead of thyroid to combat the cachexia following thyroidectomy in dogs. These observations are in part contradictory, and no one is yet in position to dogmatize on the subject. But I should certainly use adrenal and should think of thyroid in any case of thymic asthma I might be called to. I wish some of the experimentalists among our Fellows would work out this subject. I have elsewhere suggested that a double function for the thymus, and the presence as secretions or in the glandular parenchyma of antagonistic substances, one resembling adrenal and opposed by thyroid, the other resembling thyroid and opposed by adrenal, seemed to be demonstrated. A disturbance of thymic balance, uncompensated by the normal physiological antagonisms, may therefore present conditions and call for correspondingly different treatment.

Dr. Musser closes: I was so impressed at the time with the evidence of mediastinal pressure brought about by two things, the changes in the lungs as well as the very large size of the thymus gland, that I have felt since that should I see a similar case I would want to have some surgical means resorted to, unless I could be satisfied with the measure which Dr. Cohen has spoken of. I talked to Dr. Keen that evening as to the possibility of surgical relief, because it seemed to me that while, of course, shock was the dominating feature, we could have relieved that child's breathing. There was not hurried respiration from the filling up of the lungs, but breathing due to mediastinal pressure. It seemed to me that if we could have given surgical relief by opening the thorax or sternum that it would have been of the greatest amount of service to the patient. Since having seen the case, Dr. Ullom has found reports of cases that have been relieved by such measures.

Dr. Ullom closes: I have nothing to say, except that in the two cases reported the thorax was opened and the thymus stitched to the cervical fascia. There was relief of symptoms, and the case went on to speedy recovery.

POINTS OF RESEMBLANCE BETWEEN PARALYSIS AGITANS AND ARTHRITIS DEFORMANS.[1]

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A LITTLE more than a year ago I exhibited before the Philadelphia County Medical Society, in the discussion of a paper by Dr. Riesman, a case of what I believed to be rigidity of the spinal column with a tremor of only the right upper limb, like that occurring in paralysis agitans. The case was remarkable in certain of its features, and the diagnosis was difficult. No one could dispute that the tremor was very suggestive of paralysis agitans; but it was not easy to determine whether the rigidity of the spinal column, the kyphosis of the cervicothoracic region, the scoliosis of the upper thoracic spine, the deformity of the hands, like that of arthritis deformans, the crepitation of the knee-joints, the muscular atrophy, the talipes equinovarus and equinocavus, were to be attributed to the paralysis agitans or not. Extreme paralysis of the lower limbs, like that occurring in this case, is uncommon in paralysis agitans but not unknown. A brief reference has been made to this case by Dr. Pearce.[2] The patient was under my observation during many months, and died in the service of Dr. Mills, to whom I am indebted for the pathological material.

The notes of the case are as follows:

S. H., aged 59 years, was admitted to the Philadelphia Hospital, May 27, 1901. The family history was unimportant.

[1] Read March 2, 1904.

[2] Philadelphia Medical Journal, May 25, 1901, p. 1017.

She had been twice married, and had had three miscarriages.

About two years before admission she began to have disturbance of gait, her legs became weak, and she had some pain, and was obliged to use crutches. About one year later she became unable to walk at all. Twelve years before admission her neck became rigid, and she had been unable to straighten her neck since that time. She said the rigidity occurred after she had washed her hair and sat in a draught to allow it to dry. The following morning the back of the neck was swollen and her head was bent forward. She had not suffered any pain from this rigidity of the neck. It is questionable whether the patient's recollection of an event occurring twelve years previously was reliable.

At the time of admission both lower limbs were swollen from the knees downward, and she had considerable pain in the right knee. Both were tender to touch. The left leg below the knee was very much swollen, especially about the ankle. The swelling and pain on handling probably were caused by œdema. The grip was very weak in the right hand, and this condition had been present since the legs were affected. The disease had developed gradually. There was no history of a stroke. Her eyes were normal. The knee-jerks appeared to be absent. The mental condition seemed to be fairly good.

The patient came into my service June 1, 1901, and on June 22, 1901, the following notes were dictated by me.

The lower limbs are atrophied, the atrophy being conspicuous in the thigh of each side, but is masked by œdema in the leg below the knee on each side, the œdema being especially marked in the left leg below the knee. Distinct pitting on pressure is obtained in the whole left leg below the knee, but on the right side only in the foot. The lower limbs are very weak. The patient can draw up her lower limbs at the hips, and can flex them at the knees; but the movements are much restricted in all parts of the limbs. Passive movements at the right hip are not so free as at the left hip, although there is no ankylosis. Considerable crepitation is obtained in each knee. The right knee is much enlarged and presents the appearance of an old joint affection. The left knee is not normal in appearance, but chronic arthritis is not apparent here. The right thigh in its middle portion measures 12½ inches; the left 13½ inches. Movement of the toes of each foot is very slight. Resistance to passive movements in both lower limbs is very feeble. The patellar reflex on the right side is not obtained, but it is difficult to get the patient to reinforce; on the left side the patellar reflex is very feeble, and detectable only by slight contraction of the quadriceps muscle. Babinski's reflex is not obtained on either side, the movement being neither flexion nor extension.

Sensation for touch and pain in the lower limbs is normal.

No fibrillary tremors are present in the lower limbs. The patient is unable to stand.

Upper Limbs. The fingers of each hand at the metacarpophalangeal articulations are deflected toward the ulnar side. Marked flexor contractures are observed at the metacarpophalangeal joints, but the fingers are hyperextended at the articulations of the second with the third phalanges. It is possible that a mistake was made in recording this note, and that the hyperextension occurred at the articulations of the second with the proximal phalanges.

Fig. 1.

The photograph was taken after the death of the patient. The deformity of the neck is very slightly exaggerated. The back is remarkably straight, and a gradual bending forward, such as is not uncommon in paralysis agitans, is not seen. (Photograph taken by Dr. H. R. Alburger.)

The condition is such as occurs in arthritis deformans. The upper limbs and trunk are very much atrophied. Voluntary movements and resistance to passive movements of the upper limbs are proportional to the degree of muscular development.

Sensation for touch and pain in the upper limbs and over the trunk is normal. The patient has a coarse tremor involving all parts of the right

upper limb. This tremor sometimes ceases when the limb is at rest, but not always, and at times the tremor is partially arrested by voluntary movement of the hand. This tremor is suggestive of Parkinson's disease. There are no fibrillary tremors in the upper limbs or trunk.

The patient is unable to sit up in bed unassisted. The head inclines forward and to the left, and there is marked kyphosis at the cervicothoracic junction. The neck can be partially straightened, but not fully, as the resistance in the neck is too great. Voluntary movement of the spinal column is greatly restricted in all directions. No involvement of any of the cranial nerves is detected.

Urinary examination made June 15, 1901, gave the following results:

Specific gravity 1022; few pale granular casts; pus cells; trace of albumin; no sugar.

Very little change occurred in the patient's condition. She died at 3 A. M., January 27, 1903.

The necropsy was made by Dr. A. E. Ellis, January 27, 1903, at 11.30 A.M. His notes, abbreviated, are as follows:

Pathological Diagnosis. Œdema and infarction of lungs; atheroma of the aorta, chronic mitral endocarditis, fatty infiltration of the heart, and cloudy swelling, acute parenchymatous nephritis, fatty liver.

The vertebral column shows decided kyphosis, the most prominent part being about the cervicothoracic junction; also some scoliosis, the column deviating to the right, involving the upper two-thirds of the dorsal region and being most marked about the middle of this area, where there is a deviation of about 2 cm. from the mid-line. Some atrophy of the muscles of the right shoulder is seen, especially of the spinati, and there is probably some slight atrophy of the corresponding muscles of the left shoulder. There is also rigidity of the right shoulder, and possibly some restriction of motion of the left shoulder-joint also. Apparently there is free motion of elbow and hip-joint. The fingers of both hands show contractures, which are more marked in the right hand. Flexion is most marked at metacarpophalangeal articulations. There is marked ulnar deviation of the fingers, slightly greater in the right hand, from metacarpophalangeal articulations.

The knees have probably more rigidity than that due to rigor mortis. The feet are in the position of equinovarus, and equinocavus is decidedly greater on the right side.

The portions removed of the vertebræ show no ankylosis. The bodies of the vertebræ under the point of greatest kyphosis are more rigidly united than normal. This rigidity, as far as can be determined, involves about four of the vertebræ.

The brain presents no macroscopic abnormalities.

A microscopic examination of the cervical region of the spinal cord and of the anterior and posterior cervical roots made by me showed no pronounced pathological changes that could explain the tremor of the right upper limb.

Had there been no tremor of the right upper limb typical of paralysis agitans I think most physicians would have accepted the diagnosis of arthritis deformans, and yet so striking was the tremor that some who saw the case pronounced it one of paralysis agitans without any reference to arthritis deformans. I believed that this patient probably had paralysis agitans associated with arthritis deformans, but it seemed to me at least worthy of consideration that the tremor might be caused by irritation of the motor cervical roots by disease of the vertebræ. Goldscheider[1] has reported a case in which violent contractions in the muscles of the arm and forearm, shoulder and breast, seemed to be caused by anterior meningomyelitis, extending from the fourth cervical to the first thoracic segment. The irritation of the anterior roots was supposed to have caused the contractions. I may refer in this connection, also, to a case of tumor of the cerebral peduncle reported by Blocq and Marinesco,[2] which caused a tremor of the left hand like that of paralysis agitans. This case showed that tremor, even though typical, is not an absolutely reliable sign of paralysis agitans. The general appearance of my patient was so strikingly like that of paralysis agitans that this disease was believed by most of those who examined her to be the cause of the tremor.

Is it possible that the whole symptom-complex was that of paralysis agitans, and that arthritis deformans was not present in the case? If so, then paralysis agitans is capable of causing certain conditions much like those of arthritis deformans, but this resemblance between the two diseases has received scarcely any attention from physicians. It may be that a study of the two diseases in their points of resemblance to one another may throw light on each. The pathology of each is most obscure. Both have been regarded as of nervous origin, but in neither has this origin been

[1] Berliner klin. Wochenschrift, 1891, p. 935.
[2] Mémoires de la Soc. de Biologie, 1893, p. 105.

satisfactorily demonstrated. Many years ago the elder J. K. Mitchell advanced the theory of a nervous origin of arthritis deformans.

Recent investigations have shown very distinct degenerative changes in the muscles in paralysis agitans, to which I shall refer presently, and it is probable that in arthritis deformans the muscles are affected simultaneously with the joints, possibly even in advance of the joint disease.

Rigidity of the spinal column, caused by rigidity of the muscles of the back, occurs in paralysis agitans in association with rigidity of the muscles elsewhere in the body, but may we expect deformities of the vertebral column in paralysis agitans ?

J. A. Sicard and L. Alquier,[1] who have studied the deviations of the vertebral column in paralysis agitans, remark that in the recent as well as in the older works, from the time of the first article by Parkinson, in 1817, to that of Grasset and Rauzier (*Traité de médecine*, Brouardel et Gilbert, t. x.) no mention is made of deformities of the spinal column caused by paralysis agitans. The attitude in which the body and head incline forward has been described frequently.

Sicard and Alquier have found pronounced deviations of the vertebral column in twelve of the seventeen cases of paralysis agitans they studied, and in some the deformity was very great, as shown by their photographs. They believe that these abnormalities of the spinal column depend upon the muscular rigidity, and develop as the muscular rigidity develops. The deformity may be kyphosis, scoliosis, or lordosis, or a combination of these.

None of their cases were with necropsy, and in many of them, if we may judge from the photographs accompanying their article, the deformity consisted of a gradual bowing of the vertebral column, but in some of the photographs scoliosis is shown.

The most common cause of rigidity of the spinal column, according to Oppenheim, is arthritis deformans. This may implicate the entire vertebral column, or only the cervical portion, so that the head is flexed upon the breast. Oppenheim[2] thinks we shall do wisely to classify all the cases of rigidity of the spinal column

[1] Nouvelle Iconographie de la Salpêtrière, 1902, vol. xv. p. 377.

[2] Lehrbuch der Nervenkrankheiten. Third edition, pp. 274 and 275.

under chronic articular rheumatism, gout, or arthritis deformans, and this is a view held by many.

Rigidity of the back caused by muscular disease has recently received attention from E. Barg,[1] and he is able to refer to cases of this type reported by Beer, Popoff, Schlesinger, Zenner, Cassirer, and Senator. Cassirer[2] refers to cases in which disease of the muscles of the back seemed to usher in the disease of the vertebral column.

In the Strümpell-Marie type the process is confined to the vertebral column and the large joints; pain is unimportant, and the vertebral column is straight, except that kyphosis of the cervical part occurs. This is similar to the condition in my case. S. H. had a remarkably straight spine, except in the cervicothoracic region, where a prominent kyphosis was present; the shoulders, at least the right, were supposed to be ankylosed, but it is probable that the restriction of movement was caused by muscular rigidity. No ankylosis of the vetebræ could be detected at the necropsy, and under the kyphosis the bodies of the vertebræ were a little more rigid than normal, and yet not positively ankylosed. We were not permitted to remove the vertebræ from the cadaver.

Was, therefore, the remarkable kyphosis of the cervicothoracic spine in the case of S. H. the result of the rigidity of the muscles of the back and neck, which Sicard and Alquier have shown may produce very intense deformity of the vertebral column in paralysis agitans; and was the slight rigidity of the lower cervical and upper thoracic vertebræ that we detected at the necropsy also the result of this muscular rigidity? The question cannot be positively answered, but I believe it should be answered in the affirmative. Clinically the kyphosis and scoliosis were such as occur in arthritis deformans. I have referred to the fact that some investigators believe that vertebral disease may be secondary to muscular disease.

Few authors describe the ulnar deviation of the fingers in paralysis agitans. Williamson,[3] in his recent monograph on this disease,

[1] Zeitschrift für klin. Med., 1903, vol. l. p. 304.
[2] Berliner klin. Wochenschrift, 1902, Nos. 10 and 11.
[3] Sherratt and Hughes, On Paralysis Agitans. Manchester, 1901.

says: "The fingers are near to each other, and sometimes the terminal phalanges of the fingers are flexed, more or less, on the second phalanges, and the second phalanges hyperextended on the proximal phalanges, as in rheumatoid arthritis. Also the fingers sometimes deviate toward the ulnar side of the hand."

Gowers,[1] in a footnote, remarks: "In rare cases the digits deviate toward the ulnar side, as in chronic rheumatoid arthritis."

FIG. 2.

Photograph of the hands in a typical case of paralysis agitans, with ulnar deviation of the fingers only in the hand in which tremor was observed.

In S. H. the ulnar deviation and contractures of the fingers were exceedingly like those occurring in arthritis deformans, but there is the evidence of a few observers that such conditions may occur in paralysis agitans. I have been able within the past year to study a case of unilateral paralysis agitans, and it has been to me of great interest to note that the ulnar deviation of the fingers occurred only

[1] A Manual of Diseases of the Nervous System, vol. ii. p. 644. J. & A. Churchill, London, 1893.

in the hand showing the characteristic tremor of the disease. In old cases of paralysis agitans I have found deformities of the hands like those of arthritis deformans so frequently that I cannot regard them as an evidence of the association of the two diseases.

The crepitation of the knee-joints does not fully establish rheumatic disease of these joints, and the muscular atrophy, though unusual in paralysis agitans in my experience, might be explained by the long confinement of the patient to her chair and bed, for it was not intense. The deformities of the feet may be regarded as a sign of paralysis agitans as truly as those of the hands, although they do not appear to have been described.

The rigidity of the spinal column, the kyphosis of the cervico-thoracic region, the scoliosis of the upper thoracic spine, the deformities of the hands and feet, the crepitation of the knee-joints, and the muscular atrophy, do not prove that the paralysis agitans was complicated by arthritis deformans, but force upon us the question whether there be not some relation between these two mysterious diseases. I wish to be understood as advancing this hypothesis with great caution. It is my desire merely to call attention to this subject, so that further study be devoted to it.

Recently Schiefferdecker[1] has found degeneration of the muscle fibres within the muscle spindles in a case of paralysis agitans. The muscle spindles are probably concerned in the recognition of the degree of contraction of the muscles; therefore, alteration of these organs probably causes uncertainty regarding the muscular contraction and interference with the reflexes, and consequently uncertainty in the voluntary use of the muscles. The nerves within the muscle spindles and within the surrounding muscles were normal, and consequently the alteration of the muscle fibres probably was primary. Schiefferdecker regards the degenerative changes of the muscle spindles as of great importance in explaining paralysis agitans. He has found also what he regards as a specific degeneration of the muscles in which were the degenerated muscle spindles. This degeneration was not seen in all parts of the muscles. It began in the sarcoplasm.

These investigations give renewed interest to the study of the

[1] Deutsche Zeitschrift für Nervenheilkunde, vol. xxv., Nos. 1–4.

muscles in paralysis agitans, and yet Schiefferdecker is not hasty in his conclusions, and believes that some cause still unknown produces in paralysis agitans changes in the nervous and muscular systems. It may be that some similar cause produces the changes of arthritis deformans, in which the muscles, as well as the joints, are affected, and attention should be directed to the condition of the muscle spindles and of the voluntary muscles in arthritis deformans.

It is interesting in this connection to recall that in progressive muscular dystrophy, a disease believed to be of muscular origin, the bones do not always escape.

DISCUSSION.

Dr. S. Weir Mitchell: I have listened with a great deal of interest to this most valuable paper. I think I can recall cases in which it was difficult to decide to which disease to assign the symptoms. There is this to be said, however, that while you see in late paralysis agitans some of the symptoms which belong to arthritis deformans, in the latter malady you may see, I think, extreme examples of difficulty and uncertainty in movement, with absolutely no tremor. I have such a case now in mind. This is the only remark of any importance which I wish to contribute to this discussion.

Dr. James Tyson: It is well recognized that there are cases of paralysis agitans which present all the symptoms of the disease except tremor, and therefore, named *paralysis agitans sine agitatione*, just as we have an *angina pectoris* sine dolore.

Dr. A. A. Eshner: I merely want to emphasize what I understood Dr. Tyson to say, that in some cases of paralysis agitans tremor is at times, at least, though perhaps not permanently absent. I have placed on record a case, and I can recall at least one other, in which tremor was at times entirely absent, and the patient presented merely the characteristic facies and attitude and a certain amount of rigidity. I confess, however, that the existence of arthritis deformans did not suggest itself.

Dr. W. G. Spiller: The suggestion that Dr. Mitchell has made has occurred to me, also. I think no one would say that tremor occurs in arthritis deformans; but in a certain number of cases of paralysis agitans there is no tremor.

In my paper I have carefully avoided anything which would indicate that I believed the two diseases to be identical. I avoided saying resemblance of arthritis deformans to paralysis agitans, and have said "points of resemblance" to show that they are not identical. I have endeavored to point out that there is a similarity which may be greater than we have supposed.

A CASE OF SPLENOMEGALY WITH BLOOD CHANGES AND SYMPTOMS RESEMBLING THOSE OF BANTI'S DISEASE, APPARENTLY DUE TO MALARIA.[1]

By SOLOMON SOLIS COHEN, M.D.,

AND

RANDLE C. ROSENBERGER, M.D.

After Osler's careful studies[2] of conditions resembling the one about to be described, and the work of Lippy, Wentworth, and others, cited by him, an historical or pathological discussion to which no new fact could be contributed would be out of place. As the patient whose case I wish to put on record is living and apparently improving, and as the affection with which she suffers is usually chronic, there is no reason to wait for an autopsy (which we are bound to hope will long be postponed) before reporting the clinical observation. Moreover, single cases may be of importance in the final classification of a subject as yet very obscure.

As Osler has clearly pointed out, and as we can all recall as we pass in review our own experience—either of cases carefully studied and recorded, or of cases vaguely recalled of which no scientific record was made—the combination of enlarged spleen, enlarged liver, and blood changes of an anæmic or chlorotic type is not exceptional, and combinations of two of these three factors not infrequent. There seems, however, to be among the motley groups in which these pathological factors are commingled in various sequence and degree one which may be separated as possessing cer-

[1] Paper read and patient presented, March 2, 1904.

[2] American Journal of the Medical Sciences, 1900, vol. cxix.; Transactions of the Association of American Physicians, second paper, 1902, vol. xvii. pp. 428, 456.

tain additional characteristic symptoms and of which Lichty,[1] in a recent study of the literature, could find only 34 cases sufficiently described. To this group of cases Osler would affix and restrict the designation *anæmia splenica*, and even *anæmia splenica chronica*. Of its earliest stages little is, or from its nature is likely to be, known. Of its intermediate stages much has been written, and to its late stages the name of Banti's disease has been given. If we accept Osler's definition in its entirety, the case to be reported does not fall within the group he has erected, for he insists on an unknown etiology, especially excluding malaria and syphilis. On the other hand, while there is no diminution of leukocytes, the enormous size of the spleen, the peculiar pigmentation of the skin, the pulmonary, nasal, and gastrointestinal hemorrhages, the apparently secondary character of the hepatic involvement, the diminution of erythrocytes, and the low hæmoglobin content of the blood, tally precisely with Osler's description.

As the blood had been carefully studied by competent observers without finding parasites until, following the administration of quinine, it was again examined by Dr. Rosenberger—who succeeded in demonstrating an organism of a type not common, at least in the United States—several interesting diagnostic queries arise. In the absence of the discovery of the organism, would the case have come into Osler's group, and is it now to be excluded? Is it possible that in any large number of the recorded cases examination at an earlier stage—for our case is probably of not more than two years' duration—would have revealed malarial parasites? Is it possible that in recorded cases in which the parasite is said to have been absent, and in which no distinct history of malaria was given, administration of a sufficiently active salt of quinine would have driven parasites into the peripheral circulation, where they could have been found? Is it possible that some cases may depend on a parasite of the malarial group that is to be found only in the spleen? I can have no definite answer to propose to these queries, and I have not studied all the recorded cases with sufficient critical minuteness to hazard a guess. I am satisfied that in very many cases, certainly

[1] Journal of the American Medical Association, February 20, 1904, p. 528.

in those in which Osler reports its absence, malaria was not present. But are we to be restricted entirely to an etiological diagnosis in this obscure condition, as we are properly restricting ourselves wherever possible? Are we at all sure that all the accepted cases have one and the same etiology? If we may include cases of varied etiology, why draw the line at malaria? except it be on the same principle as that of Dickens' barber, who remarked to a man whom he had declined to shave, that "The line 'ad to be drawn somewhere, and he drew it at coal 'eavers." In Lichty's tables the line is not drawn at the humble plasmodium or even at less respectable germs. He includes 7 cases of malaria and 2 of syphilis. I quote in summary the other features of his analysis. Twenty-six cases were males, 8 females; the average age was thirty-six years. Melanoderma was present in 17 cases. Splenic enlargement was the most prominent feature in all, and anæmia the next. Hemorrhage occurred in 24. Hæmoglobin averaged 47 per cent.; the red cells, 3,293,000; white cells, 5594. Excluding 2 cases in which there was leukocytosis, the leukocytes averaged 4381. The liver was enlarged in 16 cases, small in 1. Ascites was present in 7, swelling of the feet and legs in 9, glandular enlargement in 3, gastrointestinal symptoms in 8, and cardiac murmurs, generally hæmic, in 11. In only 2 cases was there albumin, a trace, while the 1 reported by Lichty had considerable at times, as well as pus and casts. Of 25 cases treated medically, only 5 recovered, 12 were improved, and 7 died, hemorrhage having been the commonest cause. Operation was performed in 8 cases, with 5 recoveries and 3 deaths.

I may also note at this point that even in Osler's own series of 18 cases, malaria had been present in 5. Osler excludes it as a factor in the splenic anæmia, however, on the ground of time. In 3 of the patients (aged forty years, fifty-six years, and thirty years) the infection was in childhood. In only 2 was it in adult life; in 1, twelve years, in the other eighteen years before the onset of the present illness. In view of the tendency of the malarial organism to lie dormant for long periods, possibly in the spleen, possibly in the bone-marrow, possibly elsewhere, in an unknown stage, and over many years—more than twenty years in 1 of my cases—can we be sure that this admitted infection was devoid of moment in the cases

reported? It is true that in cases of prolonged malarial infection—and such was the fact in the instance referred to—paroxysms of various kinds are usually manifested with some kind of periodicity, recurring annually or semi-annually, as a rule, and that well-timed and repeated search in the peripheral blood, especially if the patient be placed under the influence of quinine, iron, or residence at an altitude, will find the organisms. Nevertheless, there may be, as there was in the chronic case just cited, long periods, of six months or more, during which careful study of the blood fails to find the parasites.

Thus the possibility of the existence of long-continued malarial infection, despite the failure of good observers to discover the organism, must be admitted; and with this admission, until a definite etiology—specific or non-specific—is established for the anæmia splenica of Osler, cases conforming clinically to the type may usefully be studied together. Osler's definition is as follows: "A chronic affection, probably an intoxication of unknown origin, characterized by a progressive enlargement of the spleen, which cannot be correlated with any known cause, as malaria, leukæmia, syphilis, cirrhosis of the liver, etc. (primary splenomegaly): anæmia of a secondary or chlorotic type (leukopenia), a marked tendency to hemorrhage, particularly from the stomach, and in many cases a terminal stage with cirrhosis of the liver, jaundice, and ascites (Banti's disease)."

The case history of my patient, condensed from Dr. Slifer's ward notes, may be given briefly:

SUMMARY. History of chills and fever in Italy two and a half years ago. Irregular fever, with cough etc., dependent upon acute bronchopneumonia, possibly influenzal; hæmic murmur; greatly enlarged spleen and liver; melanoderma; nasal, pulmonary, and gastrointestinal hemorrhages; marked anæmia with low hæmoglobin content; variable leukocyte count; malarial organisms not found until after cinchonization.

Maria B., the patient herewith exhibited, is an Italian girl said to be fourteen years of age, but apparently older. As she cannot speak English and an intelligent interpreter was lacking, little could be gleaned in the way of history or subjective symptoms when she was admitted to the Philadelphia General Hospital on December 12, 1903. She had at that time a slight fever, with pain in the chest and cough, accompanied with expectoration of blood-tinged mucus, and physical examination revealed signs—scattered areas of

dulness, harsh breathing, subcrepitant rales, and friction sounds—that warranted a diagnosis of bronchopneumonia, with concomitant pleurisy, probably influenzal. The Widal reaction was not manifested in the urine; tubercle bacilli were absent from the sputum, which contained pneumococci, staphylococci, and streptococci. Enlargement of the spleen and liver were noted at the time, as well as the presence of a faint systolic murmur, both at the base and at the apex of the heart. Malarial organisms were looked for and not found. The course toward recovery (of the acute attack) was uneventful. As cough, pain, and fever subsided, attention was given to the chronic conditions and the outline of spleen and liver carefully delimited by palpation and percussion. The lower border of the spleen was found to extend two inches to the right of the median line and three inches below the umbilicus. Thence the right border ran in a diagonal line toward the costal margin, which it cut one and a half inches to the right of the left mammillary line. Above the costal border percussion flatness extended to the fifth interspace in the mammillary line, to the sixth in the midaxillary line, and in the posterior axillary line to the lower border of the scapula in rest. The upper posterior corner of the splenic flatness was on the line of the upper border of the sixth dorsal vertebra, one inch to the left of the middle line of the back; thence the line extended diagonally forward to the posterior axillary line, which it cut at the margin of the ribs, there merging into the posterior border of the abdominal mass as outlined by percussion and palpation. The liver was found to reach to the eighth dorsal spine superiorly, inferiorly to the twelfth. Anteriorly the liver flatness in the mammillary line reached from the upper border of the fifth rib to a line one and a half inches below the costal margin. The left lobe was found by palpation to extend across the epigastrium as far as the right or anterior border of the spleen, when it apparently sank below the latter organ, which could be grasped separately. The abdomen as a whole was somewhat distended, the superficial veins well marked; there was no ascites; the conjunctivæ were slightly icteroid. The patient, as may be noted, is of dark skin, but has a deep red—not cyanotic—flush on both cheeks; the lips are of a good red color. Small pigmented areas are found over the entire body—the face, the neck, the thorax, the back, the arms, the abdomen, the legs, the soles of the feet. They vary in size from a pinhead to an irregular oval, whose long diameter may be one-half inch, and in hue from light yellow almost to black. The prevailing shade is a deep brown.

On admission a history of constipation was given, but this symptom was absent while in the hospital; the bowel movements on admission being, indeed, liquid and of a light color.

January 3, 1904. The patient had a slight hemorrhage from the nose, which was followed by hæmoptysis, cough, and expectoration of blood, mostly bright red and frothy, though some clots were found. She did not complain of pain. Following the hemorrhage the pulse was rapid and

rather weak and the area of splenic dulness seemed to be perhaps slightly decreased. Five days later the patient coughed up during the night about eight ounces of blood of a bright-red color, with a few small clots. In the morning epistaxis occurred, and the following night there was vomiting of about three ounces of clotted blood.

On January 10th the patient passed by bowel about six ounces of bloody fluid, described in the ward notes as "mostly pure blood, liquid and dark in color."

Examinations of the urine and of blood from the finger were made from time to time, some of which may be quoted.

December 19, 1903. The urine was amber in color; acid in reaction; specific gravity, 1016; sugar absent; trace of albumin present; no casts found.

January 21, 1904. The urine was reddish-brown in color; acid in reaction; specific gravity, 1014; sugar absent; albumin absent; no blood; no casts.

December 12, 1903. Hæmoglobin, 59 per cent.; red blood cells, 1,890,000; white blood cells, 23,450 (acute bronchopneumonia in active progress). Differential count of 200 leukocytes: small lymphocytes, 13.5 per cent.; large lymphocytes, 8.5 per cent.; polymorphonuclears, 77.5 per cent.; basophile, 0.5 per cent. An occasional megaloblast was observed; no malarial parasites demonstrable (Dr. Calhoun).

January 1, 1904. Hæmoglobin, 63 per cent.; red blood cells, 2,290,000; white blood cells, 14,000 (acute bronchopneumonia subsiding); no malarial parasites (Dr. J. C. Da Costa, Jr.). Widal's reaction, using dilution of 1 : 40, was negative in thirty minutes (Dr. Calhoun).

Failing to find malarial organisms it was decided nevertheless to begin treatment with the double hydrochlorate of quinine and urea, with the hope that this might perhaps disturb existing conditions in such a way that parasites might be found peripherally in accordance with an old clinical observation that in cases of latent malarial infection removal to a mountainous district or the administration and withdrawal of quinine would in many cases cause the development of frank paroxysms of ague.[1] Whether for this reason or for some other, Dr. Rosenberger's observation now detected the malarial organism.

January 14*th*. Hæmoglobin, 34 per cent. (following hemorrhage); red blood cells, 1,280,000; white blood cells, 10,400; nucleated reds present (Dr. Calhoun). Differential count of whites: polynuclear, 90 per cent.; lymphocytes, 6.5; hyaline, 1.5; eosinophiles, 2 per cent.; microcytes, macrocytes, poikilocytes present; also a few macroblasts, normoblasts, and phantom reds; parasites found (Dr. Rosenberger).

22*d*. Hæmoglobin, 46 per cent.; red blood cells, 2,640,000; white blood cells, 7420 (after subsidence of acute bronchopneumonia); parasites found.

[1] I first read of this fact in A. Billings' First Principles of Medicine, London, 1838, p. 218.

26th. Hæmoglobin, 48 per cent.; red blood cells, 2,690,000; white blood cells, 7800; nucleated reds present (Dr. Calhoun); parasites found.

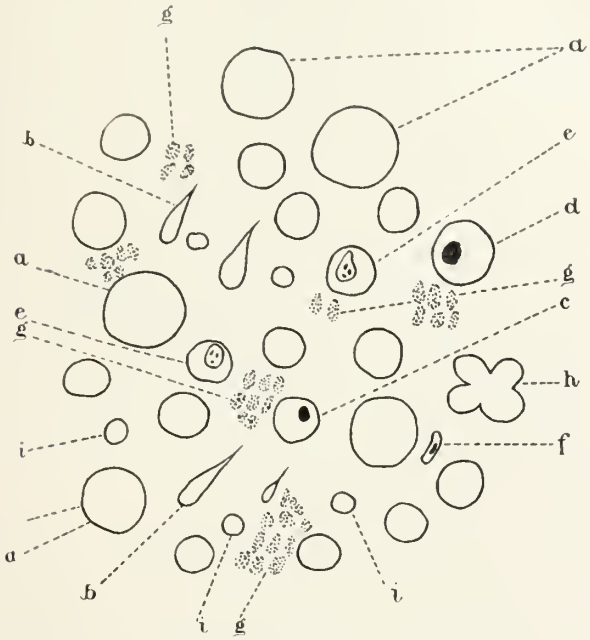
The further description of the blood will be found in Dr. Rosenberger's report.

It will be noted that the leukocyte count decreased progressively as the acute bronchopneumonia subsided; and that the last count (6600) probably represents the ordinary condition.

Quinine having been withdrawn, treatment with arsenic was begun, Fowler's solution being pushed apparently to the point of tolerance, as manifested by œdema of the eyelids and face. The dose, however, at which this symptom occurred was very small, about ten minims three times daily. The arsenic was intermitted for a week, and then resumed, when after a few days of small doses the recurrence of œdema was noticed. Arsenic was then withdrawn entirely, and remembering an old observation by J. M. Da Costa on the effect of ergot in diminishing malarial spleens, this drug was given in ascending doses, beginning with ten drops of the fluid extract and reaching thirty drops three times daily. Œdema, however, continued to recur and disappear during the administration of ergot, and it was then concluded that this was a phenomenon of the case itself, having no relation to the administration of the drug. To test the point the ergot was suspended and arsenic again given, the œdema continuing to appear and disappear during the persistent administration of arsenic and during the withdrawal of arsenic. As the hemorrhages ceased and the patient began to gain in strength an *x*-ray picture was made. The patient was not at all incommoded by the examination, and the thought was suggested of attempting to use the therapeutic influence of the rays. She was accordingly treated by Dr. Johnson with Roentgen radiations from a low-vacuum tube at a distance of twelve inches from the spleen, twice weekly; the period of exposure, at first five minutes, was gradually increased to ten minutes. In addition our object being not a therapeutic study, but an attempt to help the patient if possible, treatment with quinine and urea hydrochlorate was resumed, fifteen grains being given by hypodermic injection daily, with instruction to cease when symptoms of cinchonism should be manifested. These have not appeared up to date, but the injections have nevertheless been discontinued on account of local pain, and the drug is given by the mouth instead. W. J. Morton's observations on "internal fluorescence" by conjoining with Roentgenism appropriate medication, may be remembered here.

Whether from rest, nutrition, from *x*-rays, from ergot, from arsenic, from quinine, or from all these combined, or despite them, a great improvement has taken place in the condition of the patient. She is bright and cheerful; hemorrhages have not recurred since the date last noted, and the spleen and liver have diminished in size to the extent shown by the markings herewith exhibited, by which it will be seen, to be brief, that the outer border of the spleen has receded about two inches all around from the

previous abdominal markings and that the liver extends but seven-eighths of an inch below the border of the ribs, instead of one and a half inches as previously. Unfortunately, our therapeutic observation cannot be continued, as the Immigration Commissioners claim the girl for return to Italy as a public charge.



a. Megalocytes. *b.* Poikilocytes. *c.* Normoblasts. *d.* Megaloblasts. *e.* Intracellular parasites. *f.* Extracellular parasite suggestively crescentic. *g.* Blood platelets. *h.* Deformed erythrocyte. *i.* Microcytes.

The report of the blood examinations by Dr. Rosenberger and his comments thereon follow:

January 14*th.* Blood was thin, watery, pale, and of retarded coagulability. In the fresh state the erythrocytes were irregular in size and shape; microcytes, macrocytes, poikilocytes, nucleated forms (normoblasts and macroblasts) and a few pale or indistinct cells were present. There was no increase of white cells, and the polymorphonuclear form was the most prominent.

In the preparation were a number of small bodies, some circular, some slightly oval. Some of these bodies were slightly granular, though most were hyaline. A few contained small fine pigment granules in an amotile

state; some were intracellular, while the greater number were extracellular. Hæmoglobin, 34 per cent. (Dare's hæmoglobinometer); erythrocytes, 1,280,000; leukocytes, 10,400 (Dr. Calhoun).

Spreads were stained with Ehrlich's triple mixture, some with Wright's, and others with Jenner's stain. A differential count of the leukocytes resulted as follows: polymorphonuclear, 90 per cent.; lymphocytes, 6.5 per cent.; hyaline cells, 1.5 per cent.; eosinophiles, 2 per cent.

In these stained preparations the bodies above referred to were plainly visible, mostly extracellular and possessing a finely granular, faintly reticulated appearance. Ross' method was also used to demonstrate the parasites with success. Suggestively crescentic-shaped bodies were present.

20*th*. At this examination the blood presented the same interesting features.

In fresh preparations were the small hyaline bodies as noted in the first specimen, several of which were slightly motile.

There were few poikilocytes, an occasional nucleated red cell (normoblast) and a few phantom cells were still present, as well as the condition of microcytosis and macrocytosis. In spreads prepared with Wright's stain the bodies were evident; in some there was a background or matrix staining an extremely delicate or pale blue. Hæmoglobin, 45 per cent. (Dare's hæmoglobinometer); erythrocytes, 2,640,000; leukocytes, 7420 (Dr. Calhoun); differential count of leukocytes was: polymorphonuclear, 77.5 per cent.; lymphocytes, 14 per cent.; hyaline cells, 5.5 per cent.; eosinophiles, 2.5 per cent.; basophiles, 0.5 per cent.

In one or two hyaline cells an apparent nucleolus was situated eccentrically. Only two normoblasts were found in each of several spreads examined.

23*d*. Spreads of the blood were made at 12 M. (noon), 2 P.M., 4 P.M., and 6 P.M. In the fresh condition a few spherical hyaline, sluggish bodies were in each preparation. There seemed to be no increase in the number in succeeding examinations.

27*th*. The blood obtained upon this date differed in no respect from that taken four days previously. The bodies were clearly hyaline in the fresh blood, and in the stained spreads a few were seen intracellularly, faintly pigmented.

The irregularity in the size of the erythrocytes still persisted, while an occasional normoblast was seen. Apparent vacuolation of the red cells was also observed. Hæmoglobin, 47 per cent. (Dare's hæmoglobinometer); erythrocytes, 2,240,000; leukocytes, 6600. Differential count of the leukocytes was: polymorphonuclear, 66 per cent.; lymphocytes, 25.5 per cent.; hyaline cells, 6.5 per cent.; eosinophiles, 2 per cent. None of the leukocytes contained pigment granules.

30*th*. Spreads made on this date contained the same bodies in about the same condition as in the previous examinations, mostly oval and extra-

cellular, but a few intracellular. Microcytes and macrocytes, phantom cells, apparently vacuolated erythrocytes, and an occasional normoblast were demonstrable. A few erythrocytes were seen taking the form of a four-leafed clover.

As will be seen, our blood picture is as follows: hæmoglobin, below 50 per cent.; erythrocytes, averaging 2,000,000; leukocytes averaging between 7000 and 8000, but diminishing as an intercurrent acute infection subsides; poikilocytosis, microcytosis, and macrocytosis, presence of normoblasts, some few megaloblasts, phantom cells, and the appearance of small, oval circular intracellular and extracellular bodies, which, in small number, are very lightly pigmented.

In most respects the blood changes here recorded resemble those of Banti's disease or splenic anæmia, the chief differences being in the small number of nucleated reds found in this case and the absence of marked leukopenia. The low percentage of hæmoglobin, the small number of erythrocytes, the presence of poikilocytes, microcytes, macrocytes and phantom cells all point to this malady. Even the hæmatemesis and enterrhagia clinically suggested Banti's disease. These latter conditions may have arisen from pressure bringing about congestion as a result of enlargement of the spleen. It is possible that we may be dealing with both maladies—*i. e.*, Banti's disease and malaria.

In morphology, the small bodies while not conforming to a typical malarial parasite, may represent, as Manson says, "moribund or fragmented forms." (These are mechanically freed parasites expressed from blood corpuscles by the compression to which the blood is subjected between slip and cover glasses. Frequently, these artificially-freed parasites are broken into small fragments, and, becoming free in the liquor sanguinis, assume a spherical or disk-like form. The protoplasm becoming diffluent, the pigment is resolved into a number of minute dust-like particles possessing active brownian movement.) The intracellular forms are no doubt malarial parasites.

Golgi describes as quinine forms of the malarial parasite certain rounded, immobile bodies with clumped pigment and sharp irregular outline, and deformed segmenting bodies deficient in the number of spores.

Ewing has observed in cinchonized cases half-grown forms numerously subdivided and deficient in chromatin.

That these bodies are piroplasma (Leishman-Donovan) seems doubtful, as the latter possess a micronucleus and macronucleus, are extremely small, are rarely, if ever, situated intracellularly (red cells), and are never found in the peripheral circulation.

According to most observers, the leukocytes in malaria do not increase in number, usually remaining below 10,000, and the polynuclear cells are most numerous in differential counts made.

Lymphocytosis may also occur in some cases. In pernicious malaria as low as 40 per cent. of polynuclear cells has been found by Bastianelli.

Various changes in the erythrocytes have been noticed by many observers. Oligocythæmia, erythropyknosis, granular degeneration, as well as phantom cells, macroblasts and microblasts have been observed. Ewing states that, as a rule, in cases of moderate severity there are changes in the red cells as noted in secondary anæmia. Hæmoglobin is also lowered in percentage.

In the case herein reported there was never what might be called a marked leukocytosis, no lymphocytosis, nor was there ever evidence of pigment or the presence of any of the parasites in any of the leukocytes.

Throughout the differential counts made the polynuclear forms were most abundant. An occasional nucleolus eccentrically situated in one or two hyaline cells, and buds of protoplasm from lymphocytes were present. Occasionally a cell reacting like a plasma cell was found. The red cells in the first count were slightly less than 2,000,000; in the second and third counts there were between 2,000,000 and 3,000,000. Large clusters of small, irregularly oval granular bodies, some reacting positively to the basic dye, others to the acid dye, some intracellular and others extracellular, represented in all probability degenerating blood platelets. They were in excessive numbers.

Stained preparations are under the microscope for examination by the Fellows of the College.

DISCUSSION.

Dr. James Tyson: It seems to me that the case reported might well be called a case of malarial spleen, which differs from Banti's disease or splenic anæmia simply in that it has a cause. It is unfortunate that we are still compelled, because of our ignorance, to make classifications like that quoted by Dr. Cohen from Dr. Osler, viz., a disease characterized by an enlarged spleen, and an anæmia without blood changes which distinguish it from other anæmias, the only peculiar feature being that its cause is unknown. We know, as Dr. Cohen has told us, that many cases classified as splenic anæmia have causes, and we know wherever there are splenic enlargements there are blood changes of no distinctive character, including those which Dr. Cohen has mentioned, that is, pigmentation of the skin and changes in the size and shape of red blood cells, and so on.

It is to be hoped that ultimately all of these cases of so-called Banti's disease or splenic anæmia will be found to have cause, and that as our information grows, the number of cases which may thus be classified will diminish.

Dr. R. N. Willson: I do not think reference has been made to the fact that in this case there was no "large mononuclear" leukocytosis. This has been spoken of by McDonald as a characteristic sign in malarial infection, and in his last article it is looked upon as of great diagnostic value. In its presence he is inclined to exclude other conditions. In a case of undoubted malaria which Dr. W. E. Hughes and I studied together, the proportion of large mononuclear leukocytes was 18 per cent. Since the reading of the article previously referred to I have found in nearly every case from 10 to 12 per cent., often even 20 to 25 per cent., of large mononuclear cells. It might be an important aid in deciding whether the condition under discussion is Banti's disease pure and simple, or malaria, or the two combined. It would seem to me that it is more likely that two conditions are present, since we have the lack of the large mononuclear leukocytosis, and yet have found the supposed parasites in the blood.

Dr. Cohen closed on his part: I have only this to add, Mr. President, that I fully concur with what Dr. Tyson has said. It is worth while to put single observations on record, so that, perhaps, by multiplying the number of cases in which the signs and symptoms detailed are associated with an apparently definite etiological factor we may find that there are many etiological and pathological groups of splenic anæmias, and finally the group of unknown causation will fade out of our nomenclature. I have seen such cases in my early practice diagnosticated as malarial spleen with anæmia, and no further record made. It may be that such a diagnosis is correct. It may be that the presence of malaria is only a coincidence. Dr. Rosen-

berger's report calls attention to the point which Dr. Willson has emphasized concerning the large mononuclear lymphocytes.

Dr. Rosenberger closing on his part: I have nothing further to say, except that I decided on the diagnosis of malarial plasmodia upon the fresh blood rather than in the stained preparations. Many intracellular bodies resembling malarial plasmodia were seen, but these were not typical, and represented in all probability blood platelets.

No increase in the number of large lymphoeytes (hyaline cells) was observed in any examination.

ENORMOUS MIXED TUMOR OF THE PAROTID REGION.

REACHING TO THE CLAVICLE AND WEIGHING ABOUT SEVEN POUNDS; OPERATION; RECOVERY.[1]

By W. W. KEEN, M.D., LL.D.,
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The patient, E. M. B., aged sixty-five years, was admitted to the Jefferson Medical College Hospital January 2, 1904, at the request of Dr. H. M. Righter. Height, 5.7 feet; present weight, 152 pounds; weight in 1890, 250 pounds.

History. His father died at the age of forty-nine years of "dropsy of the brain"; his mother at fifty-six years from pulmonary tuberculosis. One sister also died of the same disease, and another after some gynecological operation; one brother died from accident; one sister is living and well. Tuberculosis exists both in his father's and in his mother's families, but he does not know the precise individuals who have suffered from it. He is married and has five children, all of whom are living. He had varioloid when he was twenty-two years old. He has suffered from hemorrhoids and from neuralgia of the face. He was discharged from the army in 1863 on account of "dropsy of the kidney." He drinks beer moderately; smokes a pipe almost constantly.

Development of the Tumor. In November, 1880, when shaving, he noticed a small lump in the right parotid region. It did not cause him any pain, nor was it tender at first. By 1883 the tumor was large enough to attract the attention of others. By 1892 it had grown to the size of a large walnut. At this time it was manipulated considerably by a physician, and from this he dates its rapid growth. He states that it has doubled in size during the last two years; has never noticed any effect on the flow of saliva. On December 7, 1903, at the inferior border, the tumor opened and there was a forcible discharge of a fluid containing blood. He states that it broke with such force as to cause a hissing sound at first. About two pints of

[1] Read April 6, 1904.

fluid escaped. For two years he has not left his house on account of the annoyance he experiences from everybody looking at him.

Examination. On admission there is noticed on the right side of the face and neck an irregular nodular tumor (Figs. 1 and 2) starting just in front of the ear, passing over the lower jaw, under the chin, and extending about 5 cm. to the left of the median line. It extends a little below the

Fig. 1

Tumor seen from the front.

level of the clavicle and passes up the back of the neck not far from the median line. The circumference of the tumor horizontally is 63 cm. and vertically 67 cm. The horizontal diameter is 21 cm. and the vertical diameter 27 cm. The present size of the tumor when the photographs were taken is markedly less than it was before it broke.

The tumor does not seem to be deeply attached to the larynx or to any of the bones in its neighborhood, the jaw, the mastoid, or the transverse

processes of the vertebræ. It is most firmly attached to the parotid region, but below this it can be swung through a considerable arc in all directions. The skin is movable over the tumor; its bloodvessels are dilated, and its surface is rough, and at points ulcerated. At the lower border is an opening about the size of a lead-pencil. Considerable fluid discharge con-

Fig. 2

Tumor seen from the side.

stantly escapes from this opening. The fluid is brownish-yellow, thin, and watery. The tumor is composed of nodular masses of greater or less size, many of the larger ones have smaller nodules arising from them. They are elastic, yet firm. Portions of the tumor are evidently cystic; others are either fibrous or cartilaginous. There are certain areas of tenderness over it. He has rarely had any pain excepting when it was distended by

fluid before its rupture. The tumor is very heavy and drags on the face and neck so much that both for support and also to retain dressings over the sinus at the lower portion, he slings it by a bandage over his head.

Examination of the Body Fluids. The urine is of amber color, turbid in appearance; specific gravity, 1026; acid reaction; urea, 2.6 per cent. By the microscope a few squamous and columnar epithelial cells and a few leukocytes are found.

Examination of the blood showed hæmoglobin, 78 per cent.; erythrocytes, 4,830,000; leukocytes, 7800; color index, 0.81.

The examination by Dr. Stellwagon of 55 c.c. of the fluid discharged from the tumor showed it to be a cloudy, yellowish-pink fluid, which, on standing, separated into two distinct layers, an upper clear and a lower cloudy stratum. The reaction was neutral to both litmus and (phenolphthalein) specific gravity 1008, that of the normal saliva being 1002 to 1006. Sulphocyanide is absent. It is highly albuminous, as shown by Tanret's reagent, heat, and nitric acid. There was also a small amount of a mucinoid substance. A very faint trace of the nitrites was present. Neither sugar nor other reducing agent was found either in the solution or the sediment. The specimen was tested for the presence of ptyalin or other amylolytic enzymes by the admixture of the solution with soluble starch; the resulting mixture was then digested for one hour at 40° C. and tested. The test for erythrodextrin and sugar proved negative. Microscopic examination showed the sediment to consist of many polymorphonuclear leukocytes and a few erythrocytes; no epithelium or salivary elements were found.

Operation, January 6, 1904. Drs. Hearn and Stewart, together with the house surgeon, Dr. Gillette, kindly assisted me. I first made an incision from the ear to the collar-bone, a little internal to the vertical axis of the tumor, then passing over it in such a direction as to give me a considerable anterior flap. This was dissected from the front and the inner side of the tumor; then, dragging the tumor outward with retractors, I was able gradually to separate it from all the deeper tissues of the neck, including the jugular vein and carotid artery. The tumor was very adherent to the vessels, the sternocleidomastoid and the auricularis magnus; a number of branches of the facial were unavoidably divided, though I did not recognize any of them. A number of large veins emptying into the jugular were found on the surface of the tumor. Each one of these was double ligated and divided. By the weight of the tumor the submaxillary gland had been drawn down over the anterior surface of the tumor to the level of the upper border of the thyroid cartilage. This was dissected off the tumor and the facial artery, which passed through it, was double ligated and divided. I then formed a posterior flap of skin similar to the anterior, and finally removed the entire tumor. At probably a dozen or fifteen places the fascia between the lobules was spread out in dense fibrous layers,

which had to be divided one after another, the division of each one enabling me to roll the tumor outward with greater ease. In the carotid region, in dissecting it out, a moderate amount of the parotid gland was removed. As there was considerable oozing over the entire surface, I placed two layers of iodoform-gauze on the surface, inside of this two layers of plain sterile gauze, and closed the entire wound, leaving the gauze protruding at the lower end.

During the operation he was given four pints of salt solution and two drachms of adrenalin (1 : 1000) gradually administered. The operation lasted an hour and ten minutes.

Blood Pressure. The blood pressure the day before the operation by the Riva-Rocci apparatus was 164 mm. When the etherization was begun the blood pressure was 210 mm. It rose within ten minutes to 230 mm., but when the first incision was made it fell to 205 mm. At the same time the pulse, which before etherization stood at 80, gradually rose during the etherization to 125, where it stood at the time of the first incision. The blood pressure rose slightly after the operation was begun, and then, as it was continued, fell to 185 mm. at the end of the first half-hour. The infusion of adrenalin and salt solution was then begun, and the observations on the blood pressure necessarily were interrupted by this procedure, as it was necessary to use the left arm for both these observations and the infusion, since I was standing on the right side and could not allow the right arm to be used. After the operation the pulse remained at the same level (125), but the blood pressure had fallen to 110 mm.

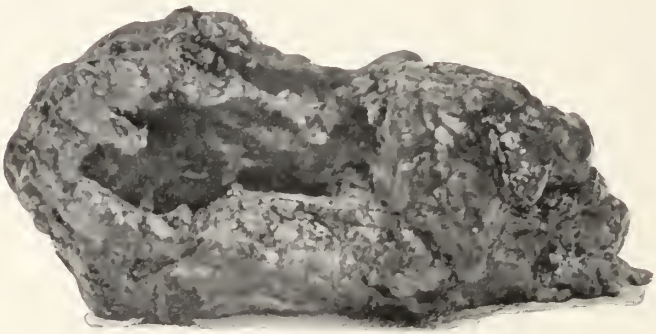
Convalescence. His highest temperature after the operation only reached once a little above 99°, until one week after the operation, when it rose once to 101°. The cause for this was perfectly clear. The incision was close to the corner of the mouth on the right side. The operation necessarily paralyzed the seventh nerve, which was involved in the tumor, and in consequence of this there was a constant dribbling of saliva. This produced a slight infection at the middle of the wound, and delayed the final healing for a considerable time. Other than this he made a perfectly smooth recovery.

Examination of the Tumor. At the time of the operation a culture was taken from the discharge through the open sinus. Professor W. M. L. Coplin reported that after forty-eight hours a pure culture of *staphylococcus pyogenes albus* was present. The specimen (Fig. 3) shows the tumor and the large cavity in the centre. The size of the tumor was 25 cm. long and 17.5 cm. in diameter. The weight was 2440 grams (over $4\frac{3}{4}$ pounds), to which, before the rupture, should be added over 2 pounds of liquid, which escaped, making the tumor originally about 7 pounds in weight. I give the remainder of the report by Professor Coplin and Dr. Ellis in full:

"*Gross Anatomy.* A long incision which has been made into the specimen, on the surface opposite the area of skin, shows it to contain an enor-

mous cavity that forms a large part of the bulk of the mass, the tissues described being in the form of a shell that varies from 1 to 8 cm. in thickness. The cavity is entirely devoid of contents. The limiting wall is gray in color, with pinkish markings, and is made exceedingly rough by projecting ridges of tissue. These ridges traverse the wall in every direction, and are from 1 to 3 cm. in width at their bases, narrowing outward until in many instances they become very thin at their most prominent parts. Owing to the curvature of the wall of the cavity the external margins of two of these ridges are, in some sites, brought in close proximity, the spaces between them leading into pocket-like recesses which closely approach the external surface of the specimen. The ridges are formed of tissue similar to that making up the exterior of the mass. The incised surfaces are gray in color and have a cartilage-like consistency. At several points the density of bone is almost or quite attained.

Fig. 3.



Tumor after removal, showing the large cavity.

"*Microscopic Examination.* Small portions of the specimen were fixed in Bensley's and Zender's fluids and in 10 per cent. formalin. The gross specimen was preserved in Kaiserling. Blocks of tissue from the former were dehydrated, cleared, and infiltrated with celloidin. Sections from these blocks were stained with the hæmatoxylin plus eosin of Van Gieson, toluidin blue, thionin, Mallory's reticulum stain, and Weigert's stain for elastic tissue.

"Histologic study of the sections shows them to be composed of very diverse structures. Those including a part of the external surface of the tumor show a thick fibrous capsule. The external portion throughout and at some points nearly the entire substance of this capsule is extremely dense and practically a vascular fibrous tissue. The inner portion is less dense, contains distinct bloodvessels, and in some areas is quite cellular in type. At certain points are small areas of adipose tissue, adjoining which are variously shaped small open spaces in the dense outer portion of the cap-

sule that appear to be included fat. Within the capsule are areas of the tumor composed of cells within a matrix of either fibrous or myxomatous tissue or areas of cartilage. The cells are mainly round or oval, with here and there a few that are spindle shaped. In some areas they form distinct masses with but scanty intercellular substance, while in others they line spaces, presumably lymph spaces, being here from one to many layers thick. As previously stated, the supporting structure in some of these areas is fibrous tissue, which in some instances forms distinct trabeculæ. In other areas the matrix is myxomatous. In certain of these areas the cells are numerous, forming quite distinct masses; in others they are comparatively scanty, being arranged in narrow bands that cross each other in such a way as to give a distinct plexiform appearance. Here the cells are oval or flattened in shape and the nuclei stain poorly, contrasting markedly with the masses of larger, round, deeply staining cells, which in many instances are separated from them only by a moderately wide band of fibrous tissue. The former present the appearance of true sarcoma cells, and have among them numerous thin-walled bloodvessels, while the latter are undoubtedly derived from the endothelial lining of the spaces which they enclose.

"Many of the sections are composed partially, in some instances almost wholly, of cartilage. This is usually fibrous in type, though areas of the hyaline variety are to be noted. The capsules of many of the cartilage cells are wrinkled and contracted. In a few areas bone formation has been attempted as shown by the presence of osteoid tissue containing lacunæ and distinct cells, but lacking true lamellæ.

"At some points the stroma between masses of the tumor cells has undergone hyaline change, giving such areas a very close resemblance to the so-called cylindromas.

"Sections which include the inner surface of the tumor, the wall of the large cavity, show it to be bordered by softened fragmented and slightly neurotic tumor tissue. A few polymorphonuclear leukocytes are also present.

"Sections stained for elastic tissue show an abundance of that structure in the stroma of the tumor, especially in the less dense areas where the cells are least numerous.

"Sections stained by carbol-thionin show in some areas an exceedingly fine network that stains a distinct reddish tinge.

"*Diagnosis.* Myxo-chondro-lymphangio-endothelioma. Though no salivary gland structure has been found in the sections studied, the specimen conforms in every particular to the mixed tumors which occasionally originate in those organs, particularly the parotid."

Remarks. The diagnosis and structure of these mixed tumors of the parotid region have been so thoroughly considered by Dr.

A. O. J. Kelly[1] and Francis Carter Wood,[2] that I do not deem it necessary to consider them here anew, as I could add nothing of importance.

Fig. 4.

The patient three months after operation.

From the surgical point of view, the slow and later the rapid growth, the enormous size (certainly the largest tumor of the neck

[1] Philadelphia Monthly Medical Journal, February, 1899.
[2] Annals of Surgery, January and February, 1904, pp. 57 and 207.

I have ever operated on), his age, and the fact that I was able to dissect it away from the important structures, especially the blood-vessels of the neck, and the very happy recovery of the patient, justify the report of such a case.

I may add that not only is he well, but, after having been practically imprisoned in the house for over two years by reason of his annoyance when people observed him, he now once more freely goes out and mingles with his fellow-creatures. The operation has given him a new lease of life. The only price that he pays for it is a long visible scar and paralysis of the right seventh nerve, a small price for such a delightful relief. Fig. 4 shows the result three months after operation.

A PRELIMINARY COMMUNICATION UPON THE RESULTS OF SOME STUDIES OF AORTIC REGURGITATION.[1]

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The physical signs of insufficiency of the aortic valves cannot be said to have been clearly understood by Laennec. In the 1819 edition of his book his description of the physical signs is vague and inconclusive. He is not clear regarding the time of the murmurs, and is quite indefinite regarding the importance of their localization. Under these circumstances it is not surprising that in Observation XLV. he describes the case of a boy who complained of palpitation of the heart, with the following physical signs: the cardiac pulsation was vigorous, there was a purring thrill alternating with the pulse, and a murmur resembling the filing of wood heard during the contraction of the auricle (the diastolic interval), synchronous with the thrill, and a louder murmur heard during the contraction of the ventricle, synchronous with the pulse. There was no cyanosis, no irregularity of the pulse, and never any œdema, the patient complaining chiefly of disturbed sleep. These physical signs would nowadays be interpreted as indicating aortic regurgitation, but Laennec made a diagnosis of ossification of the mitral valve, slight hypertrophy of the left ventricle, and perhaps a slight ossification of the aortic valves. The patient recovered as a result of repeated venesection. This subject was industriously studied for the ensuing forty years, and in 1864, when the sixth edition of Skoda's book appeared, the medical profession was in a position to make and receive definite statements regarding the value of physical signs.

[1] Read April 6, 1904.

During this interval practically all medical progress had been along the lines of physical diagnosis. Laboratory work still occupied a very subordinate place in clinical medicine. Nevertheless, the energy devoted to classification showed that progress was becoming partially arrested. According to Skoda the diagnosis is to be made from the enlargement of the heart, a very loud diastolic murmur heard best at the aortic cartilage, and often, when the insufficiency is extreme, also heard at the apex of the left ventricle, replacing the second sound, and usually a systolic murmur heard best in the aorta, and probably generated by roughness in the wall of the vessel or upon the under surface of the aortic valves. He also speaks of the rasping sounds heard in the subclavian and carotid arteries. He particularly objects to regarding the murmur of aortic insufficiency as the *bruit de va et vient*, because the systolic murmur does not depend upon the insufficiency, and the latter must not be diagnosed unless the diastolic murmur is present and should be diagnosed when the systolic murmur is absent and replaced by a sound. Stokes, who is quite as accurate an observer as Skoda, insists that the typical murmur of aortic regurgitation is the bellows murmur, that is, the to-and-fro sound. Flint, in this country in 1870, laid stress upon the importance of the diastolic, and minimized the importance of the systolic murmur. The influence of Skoda in Germany and of Flint in this country has been so great that, in spite of the fact that both authorities speak of the great frequency of the systolic murmur, modern writers are apt to disregard it almost entirely. Thus, Musser speaks of a systolic murmur at the second costal cartilage on the right, transmitted into the vessels of the neck, which is due to roughening of the valve segments or to atheroma of the aorta. This is really a reiteration of Skoda's view. Von Leube regards the diastolic as the only pathognomonic sign; when it is present aortic regurgitation should be diagnosed, and when it is absent aortic regurgitation should be excluded.

I have for some years been collecting records of heart cases and confirming them whenever possible by autopsy. This confirmation has been comparatively rare, and yet has often been of considerable value. In my experience at Blockley and the University Hospital and the out-patient department of the Howard Hospital, aortic

regurgitation has been the commonest valvular lesion. This is contrary to the established view that mitral regurgitation is by far the commonest lesion. It agrees, however, with the view of one of the earliest students of physical diagnosis, Corvisart, and it must be remembered, as Laennec remarks in the first edition of his book, that the experience of one investigator regarding a statistical subject is very fallacious. He found, for example, that his statistics at two different hospitals to which approximately the same class of cases was admitted gave very different results. Flint also speaks of the varying frequency of heart murmurs, and particularly the frequency with which he came across the murmur of mitral stenosis, at that time regarded as excessively rare. I have attempted to analyze some of the material which I have accumulated, and to formulate certain views which have grown out of my studies. This evening I shall merely read, in the form of a preliminary communication, some of the results at which I have arrived.

Since the description of the characteristic pulse by Corrigan the investigation of the peripheral circulation has been considered of importance. In no valvular disease of the heart is the same amount of alteration produced in the pulse. In about three-quarters of the cases a distinct increase in the volume of the pulse was observed when the arm was held vertically. This increase is usually so marked that it serves at once to determine the character of the lesion. Von Leube states that the *pulsus alter et celer* occurs in anæmia and in febrile conditions. I have never been able to satisfy myself that this is so, but in two cases of mitral obstruction the *pulsus alter et celer* was distinctly present. In both cases there was no enlargement of the left ventricle, the second pulmonic sound was increased, there was a typical presystolic rumble with an accentuated first sound, and the action of the heart was exceedingly irregular. One of the cases was only under observation for a week and then left the hospital. The other I persuaded to return to my office at intervals, and he has been under observation for nearly four years. During all that time, aside from the moderate alteration in the character of the pulse, there has been no sign of aortic regurgitation. Compensation frequently becomes impaired, and is usually easily restored, and there is often œdema of the feet.

In the last two years considerable improvement in the patient's condition has occurred, and he is now able to earn his living.

It has seemed to me not impossible that under certain circumstances with a simple or concentric hypertrophy of the left ventricle, such as occurs in mitral obstruction, mechanical conditions could exist that would tend to produce a quick pulse, that is, a relatively small quantity of blood would be thrown into the aorta with such force that it would produce a high wave which, on account of the small quantity of blood, would subside quickly. The characteristic shortening of the systolic and lengthening of the diastolic period of mitral obstruction were present in both cases, and may have aided in producing this result.

In no case of aortic regurgitation have I been unable to obtain the thrill in the muscles of the forearm. It varies considerable in intensity. Sometimes it is felt with slight pressure; at others it requires considerable pressure with both hands upon the muscles in order to elicit it. It has been supposed to be due to the capillary pulse in the muscles, but I am convinced that it is transmitted from the large arterial trunks of the forearm. It can be obtained only in the bicipital groove in the upper arm, and if it were due to eddies in the muscles it should be felt easily in the biceps.

The facility with which murmurs and thrills are produced in the carotids, and particularly in the subclavian arteries by slight pressure would also be in accordance with this view.

Of the auscultatory signs I have found Traube's exceedingly inconstant. Duroziez's sign is much more constant. It can only be obtained, as a rule, by very considerable pressure over the groin or over the brachial artery, and the pressure must be made chiefly with the edge of the stethoscope toward the heart. The diastolic murmur is, as a rule, exceedingly faint. In a few cases it has been quite audible, but is never nearly as loud as the systolic murmur. The latter appears always before the former, and the pressure usually must be increased considerably before the diastolic murmur is evident. Duroziez's sign is much more common in the femoral than in the brachial artery, and I have not been able to obtain it in any other arteries of the body.

A systolic murmur is sometimes heard over the skull, but in my

own experience only over the arteries of the scalp, in which it is probably generated.

Visible pulsation in the bloodvessels of the mucous membranes and of the retina is of course common, and, as a rule, easily seen. Dr. Riesman has studied the former in a very excellent paper.

The size of the heart is quite variable. In four cases the apex beat was in the fifth interspace inside the nipple line; in three of these cases the area of absolute dulness extended to the left of the left parasternal line, but in one the area of absolute dulness was normal. None of these patients presented severe symptoms of heart disease, and I infer that the opening in the aortic valves was so small that, although it produced a mechanical, it failed to produce a physiological regurgitation. Cases with severe symptoms invariably had enlarged hearts. The apex beat, excepting in the last stages, was broader and more powerful than normal. Of the thrills that were palpable the systolic was more constant than the diastolic. It is usually most distinct in the suprasternal notch or in the region between the second costal cartilage on the right and the suprasternal notch. The diastolic thrill could usually be felt over the sternum and the upper portion of the precordium, but not infrequently it was quite perceptible at the apex. In one case the diastolic thrill was transmitted upward and could be felt in the carotid arteries.

Flint remarks that: "The study of the murmurs has so much engrossed the attention of clinicians that the heart sounds have not received that attention which their importance claims. Abnormal modifications of the heart sounds afford in certain cases, as has been seen, valuable aid in the localization of murmurs. They also serve to supply in some measure information which is not afforded by the murmurs *per se*, namely, the amount of damage which the valves have sustained." It might be added that a study of the heart sounds is often of direct value in making a diagnosis. In aortic regurgitation the first sound is absent at the apex in the majority of cases. If heard at all it is usually to the left of the sternum in the third or fourth interspaces. The second sound is accentuated at the pulmonic cartilage in about half of the cases. Skoda believed that not infrequently the second aortic sound was audible and indicated that the valves were partly competent. Leube

to my mind more correctly interprets this as an accentuated second pulmonic. It is sometimes very difficult to discriminate between the accentuated pulmonic and aortic second sound. While I am not sure that the following method is invariably correct, it furnishes at least a rough test. If the accentuated second sound is transmitted downward along the left border of the sternum to the epigastrium, it is probably pulmonic in character; if it is transmitted into the vessels of the neck, and is also heard well at the apex of the heart, it is probably aortic in character. It is exceedingly rare for the accentuated second sound in aortic regurgitation to be heard in the vessels of the neck or at the apex, and therefore I am inclined to agree with von Leube. I cannot, however, agree with his further statement that the accentuated pulmonic second sound is in favor of associated mitral regurgitation, if a systolic murmur is heard at the apex.

In looking at hearts post-mortem I have been impressed by the remarkable dilatation of the first portion of the aorta, and it has occurred to me that perhaps during cardiac systole this would press upon the pulmonic artery, so increasing the resistance of the second heart that an accentuated pulmonic second sound would be present. In several cases in which the accentuated second sound existed, and upon which autopsies were obtained, there was no mitral lesion. The disappearance of the first sound at the apex is difficult to explain. The muscular element, if there is a muscular element in the first sound, which I think improbable, should be increased on account of the hypertrophy as well as the dilatation of the ventricle. The fact remains that in the great majority of cases the first sound has disappeared completely. This is not the case if a Flint murmur is present. In four cases I have heard what I regard as a true Flint murmur. Upon one of these cases an autopsy was obtained, and no mitral stenosis was present. It will be observed that my figures are considerably lower than those of Thayer. I have been impressed with the fact that there is a good deal of difference of opinion among clinicians as to what constitutes a Flint murmur. According to Flint himself it was a presystolic rumble stopping at the first sound, and resembling in all respects the murmur of mitral stenosis. Therefore, the mere existence of a murmur in the latter portion of the

diastolic interval would not be a Flint murmur in the sense in which Flint understood it, and could not give rise to any difficulty in diagnosis between aortic regurgitation and mitral stenosis.

The first diastolic murmur is by no means uncommon in aortic regurgitation, nor is it by any means uncommon in mitral stenosis in those cases in which a presystolic rumble happens (usually for a time) to be present. It is true Flint does not specifically state that the murmur terminates with an accentuated first sound, but he speaks of its sudden stoppage, which possibly means the same thing. The Flint murmur has also been heard in a variety of other conditions, particularly in dilatation of the heart secondary to obliterative pericarditis and in simple dilatation of the heart, in either case without aortic regurgitation (Phear, Murphy). Therefore, the explanation which Flint gave of the mechanism that brings about its occurrence cannot be correct.

Theoretical explanations of cardiac sounds are always of doubtful value, yet in many cases we are absolutely unable to obtain anything better. This is particularly true of the Flint murmur. It has seemed to me that the following explanation might account for its occurrence as well as any other: in aortic regurgitation, in obliterative pericarditis, and in dilatation of the heart we have an enlargement of the left ventricle. If we assume that the mitral orifice maintains its normal size it would follow that the ratio between the capacity of the left ventricle and the mitral orifice would be so altered that there would be a relative stenosis. There is no reason for supposing that this form of stenosis might not produce a murmur similar to the murmur produced by an organic stenosis in those cases in which the ventricle maintains its normal capacity and the mitral orifice is narrowed. Of course, this does not explain why a Flint murmur does not always occur in cases of aortic regurgitation with intact or at least undilated mitral orifices. But we do not know why the presystolic rumble does not occur in all cases of mitral disease.

The point of greatest intensity for the diastolic murmur varies considerably. Skoda places it at the aortic cartilage; von Leube, to the left of the sternum in the upper portion of the precordium. In the majority of cases I have observed it as loudest at the aortic

cartilage; in a very large number in the third interspace to the left of the sternum; a somewhat smaller number at the apex, and in two it was loudest at the fifth interspace on the left border of the sternum.

The systolic murmur also varies in intensity. As a rule it is heard best in the third interspace to the right of the sternum. Occasionally it is loudest in the suprasternal notch, and almost invariably it is well heard and occasionally is loudest at the apex. In one case of aortic stenosis with relative aortic regurgitation the systolic murmur was loudest at the apex, and the same thing was true in another case which was diagnosed as aortic stenosis, but upon which an autopsy was not obtained. The sphygmographic tracing also indicated some obstruction in the pulse wave. It has seemed to me likely that the murmur is normally transmitted to the apex for the following reason: At the moment of its generation the wall of the heart is rigid and therefore in a condition for transmitting well vibrations that may be imparted to it. These vibrations are received at the base and transmitted along the walls, becoming gradually intensified at the apex, where of course they are heard very distinctly.

Experience has taught me to regard with grave suspicion any murmur heard at the apex if it is also transmitted into the carotids. In one case in which a double murmur was heard distinctly at the apex and very faintly at the base, and the systolic murmur was heard very faintly in the carotids, a diagnosis was made of double mitral disease. The autopsy, however, showed aneurysm of the sinus of Valsalva, causing stenosis with insufficiency of the aortic valves. The systolic murmur is often loudly transmitted as far as the middle of the forearm, and occasionally can be heard distinctly along the course of the abdominal aorta.

In my experience the systolic murmur has been loudest in young males whose arteries have not been particularly atheromatous, and I am more inclined to regard it as caused by the dilatation of the first portion of the aorta, which is practically invariably present in all cases of aortic regurgitation. It may be the loudest murmur generated in or near the heart, and in one instance I was able to hear it with the ear six inches from the chest wall.

The differential diagnosis between aortic regurgitation and aortic regurgitation associated with mitral disease is not, in my experience, as easy as Von Leube and Thayer describe it. In one case that I have already reported there was absence of enlargement of the heart to the right and an extremely typical Corrigan pulse without marked increase in the dicrotic wave, although at the autopsy the mitral valve was found to have calcified, admitting of very free regurgitation. On the other hand, in a case with a musical murmur heard at the apex, but not at the base, with no accentuation of the second sound and no enlargement of the heart to the right, a fenestration was found in one of the mitral leaflets through which a very minute stream of blood had evidently been forced during systole. This appeared to be congenital.

I have already stated why I do not regard the accentuation of the second pulmonic sound as sufficient evidence of mitral insufficiency. The diagnosis of aortic regurgitation complicated by aortic stenosis is much more difficult. If the systolic murmur is usually produced by the dilatation of the aorta it is not necessarily indicative of aortic stenosis. In one case in which the diagnosis was successfully made it was based upon the character of the pulse, the character of the sphygmogram and the remarkable prolongation of the systolic interval. In another case the same conditions existed, but an autopsy was not obtained. The existence of relative aortic regurgitation has been frequently disputed. Many clinicians, however, recognize that it occurs, and usually state that it is produced by excessive pressure in the general circulation, such, for example, as occurs in chronic Bright's disease. I have observed it in one case, the case of aortic stenosis, to which reference has already been made. At three distinct intervals during the time this case was under observation the symptoms of aortic regurgitation appeared. At the autopsy the adhesion of two leaflets sufficed to narrow the orifice to less than half its area, and the other leaflet had apparently enlarged enough to close the opening, but any dilatation would certainly have produced insufficiency. I subsequently made sections through several aortas at their commencement, staining them with Wiegert's elastic stain and by the usual methods, and failed to find in the tissues any reason why dilatation should not readily occur at this point,

such as the existence of a dense band of fibrous connective tissue which presumably would not stretch. In fact, just at the beginning of the aorta the elastic tissue was particularly abundant.

DISCUSSION.

Dr. Frederick P. Henry: It is impossible to discuss all the details of this elaborate and interesting paper, and I will therefore allude to but one which seems to me of special interest. Dr. Sailer has referred to a case in which two of the aortic leaflets were adherent and the third much enlarged so as, to some extent, to overcome the valvular defect. Such observations suggest the possibility of cure of these lesions, and I may call attention to the fact that both Professors Leyden and Eichhorst have reported cases in illustration of this possibility. It is well known also that a lesion permitting mitral regurgitation may, in course of time, be converted into one which causes obstruction to the flow of blood from the left auricle into the left ventricle. It follows, therefore, that in the course of this transformation there is a stage when compensation is complete and the valvular defect is cured. Unfortunately, the process of sclerosis continues, and the cure is but temporary.

SURGERY OF THE PROSTATE GLAND.[1]

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The subject of the surgery of the prostate gland is one that has vexed practitioners for generations, and is even now far from being in a satisfactory state. That advances have been made in treatment, and many patients relieved of tortures worse than death itself; that operations, once almost surely followed by death, are now almost certain to succeed, that the profession is becoming more unanimous with regard to the indications for one operation rather than another in certain cases, and that an operation is no longer needlessly postponed until the patient has at least one foot in the grave—these are all signs of the ceaseless activity of the modern surgeon in searching unremittingly, conscientiously, and successfully for the greatest good obtainable for those unfortunates who come under his care suffering from the distressing effects of hypertrophy of the prostate gland.

That many patients will do well in a life of catheterism, while some will begin to fail immediately it is commenced, is a fact now well recognized, and it is the purpose of the present paper to urge an early resort to operative measures in those patients in the latter class, believing, as I do, that operation here, though necessarily an evil, is yet a less evil when undertaken early than when postponed until the last possible moment.

The rise of prostatic surgery has been well sketched by Gouley, and it is from his address that many of the following references have been obtained.

[1] Read April 6, 1904.

It appears that the ancients considered that patients with prostatic hypertrophy suffered from "excrescences" or "carnosities" at the neck of the bladder, and that when these offered obstruction to the evacuation of the bladder, their destruction was attempted with metallic instruments, introduced, of course, through the penile urethra. The existence of the prostate gland was unknown until the middle of the sixteenth century, its discovery being attributed to Nicolaus Massa.

Riolanus was the first to suggest, in the early part of the sixteenth century, that the bladder could be obstructed by a swelling of the prostate. This surgeon, in several cases of urinary retention, successfully practised incision of the neck of the bladder through the perineum, but it is not recorded whether the cause was enlargement of the prostate gland.

John Hunter, Sir Everard Home, Brodie, and others, both recommended and practised tunnelling of the obstructing body by the catheter; but this remedy was finally abandoned as dangerous. Chopart records that when Astruc, ten years before his death, which occurred in 1766, was attacked by retention of urine, Lafaye attempted to introduce a catheter, but met with an obstruction from a tumor in the neck of the bladder. He therefore perforated this by a lance-shaped stylet introduced through the catheter, which was open at the end, and by this means succeeded in forcing the catheter into the bladder and drawing off the urine. The catheter was retained fifteen days. This false passage through the obstructing body persisted, and a catheter was introduced by it as occasion required through the remaining ten years of Astruc's life, and at the post-mortem examination the condition of the parts was found to be just as described. Chopart himself tried tunnelling of the prostate several times, but with fatal results.

Systematic compression, to maintain a patulous urethra, first proposed by Physick, of Philadelphia, was employed by Leroy d'Etiolles and Mercier; but probably its best-known advocate was Mr. Reginald Harrison, of London, who employed olivary bougies, which were passed once in forty-eight hours or less often. The bougie was so shaped that the olivary swelling caused dilatation of the urethra and compression of the prostate as the instrument was passed into

the bladder, and again as it was withdrawn, it being allowed to remain in place for several minutes.

Perineal operations came into favor earlier than those by the suprapubic method, owing, probably, to the greater familiarity of surgeons with operations in the former region, due to the then widespread practice of perineal lithotomy. Covillard, in 1639, successfully operated by perineal cystotomy, and removed a hard mass, not a stone, crushing and destroying it during extraction with the forceps.[1] Chopart describes how Desault, who died in 1705, after removing a calculus by perineal lithotomy, found and twisted off a tumor in the bladder; and Sir William Blizzard, several times before 1806, performed perineal prostatotomy for enlargement without any calculous formation. Such an operation, combined with lithotomy, was by no means infrequent in the early part of the nineteenth century, and was sanctioned by Sir William Ferguson, who employed this procedure even before 1848.

Amussat removed a calculus and a protruding mass of the prostate by suprapubic cystotomy before 1832.

But the first regular surgical procedure was established in 1834 by Guthrie, under the name of "division of the bar at the neck of the bladder," this bar being in some cases due to the enlargement of the two lateral lobes of the prostate drawing up a fold of mucous membrane across the vesical orifice of the urethra. He accomplished his purpose by a catheter carrying a concealed blade. Where marked prostatic enlargement coexisted he advised perineal prostatotomy, but it is not certain that he ever performed it. Mercier, whose name is pre-eminent in the early days of prostatic surgery, devised, in 1837, special instruments—called by Gouley, prostatotome and prostatectome—and at later dates modified them in various ways. These instruments were used as the internal urethrotome is at the present time, through the penile urethra, without either a perineal or a suprapubic opening, and did not meet with very general favor. Leroy d'Etiolles, as well as Civiale, claimed priority over Mercier in the introduction of the operation since known by his name, but it appears that their claims are ill founded.

[1] Sir Henry Thompson considered the "excrescence" a true tumor of the bladder, and not part of an hypertrophied prostate; but Gouley speaks as if it were prostatic.

A further improvement on Mercier's method was that introduced about 1873 by Bottini, who aimed to avoid hemorrhage attendant upon Mercier's operation by the use of galvanocaustic apparatus. Gouley, however, states that the bleeding by Mercier's method was trifling, and that, therefore, Bottini's modification was unnecessary. Although there are occasional reports of cases treated by Bottini's method during the twenty years or more following his first description of it, yet they were by no means frequent until after the publication in 1897 of the well-known paper by Freudenberg, in which he described many improvements in the requisite apparatus. This surgeon, four years later, recommended the addition of a centimetre scale to the shaft of the Bottini cautery, in order that the operator might have a more definite idea of the position of the beak of the instrument when in use. Further modifications of the galvanocaustic apparatus have been introduced by Dr. H. H. Young, of the Johns Hopkins University; he, with Willy Meyer, of New York, and Orville Horwitz, of Philadelphia, being among the most prominent advocates at the present time of this operation, to the practical exclusion of all others.

Meanwhile, various other methods of treatment had been introduced. Of these the most important are those that arose from the practice of tapping the bladder in cases of retention of urine where passage of the catheter proved impossible. Simple catheterization to relieve the bladder of its residual urine had long been employed, and it is interesting to note that the clever manœuvre of increasing the curve of the catheter by partially withdrawing the stylet as its beak approaches the obstruction was practised and taught by Physick, the father of American surgery, long before it was accidentally discovered by Mr. Hey. Where it was impossible to introduce the catheter the bladder was punctured, either suprapubically or through the rectum. It was feared that urinary extravasation in the former situation would be more dangerous, and hence the majority of surgeons rather preferred the rectal route, it being found in many instances that the channel thus formed served fairly well for voluntary micturition (even after the cannula was withdrawn) until the urethra again became patulous by the subsidence of inflammation. Perineal puncture, though practised during the seventeenth

and eighteenth centuries, fell into disuse during the early part of the nineteenth, the rectal route being the favorite.

Thus arose the treatment by urinary fistula, and from the concomitant drainage of the bladder it may be considered a distinct advance in therapeusis. Sir Henry Thompson narrates that he saw some patients of Mr. Thomas Paget who had had their bladders punctured suprapubically, completely relieved of the tenesmus and other distressing features by wearing a cannula or a catheter in the suprapubic fistula, and that this sight gave him encouragement to try the effect of permanent suprapubic drainage, even in cases with no retention of urine. When a suitable case presented itself he accordingly introduced a long curved sound through the urethra, and, making it impinge upon the wall of the bladder above the pubic symphysis, cut down upon it with a small incision, and introduced the tube. In many cases in which he employed this method the relief afforded was marked, but he later abandoned this plan of treatment for drainage through the perineum. Dittel, Keyes, and Swinford Edwards were among the other surgeons who recommended the suprapubic fistula.

An important improvement in the method of forming the suprapubic fistula was that introduced in 1888 by Hunter McGuire. This consists, in brief, of establishing a fistula through the abdominal walls obliquely upward from the bladder, so that the fistula "bears the same relation to the bladder that the spout of the coffee-pot does to the bowl." By this procedure McGuire was able to completely relieve his patients of their cystitis and their residual urine, no involuntary leakage occurring even in the supine position, and the patients, in some instances, being able to project the stream of urine in a parabolic curve to a distance of several feet by voluntary contraction of the bladder. The urine was retained for from two to six hours. Morris, of New York, in one instance clothed his fistulous tract with skin by transferring narrow cutaneous flaps into the wound at the time of operation.

The treatment by perineal fistula developed as a natural consequence of perineal cystotomy, and was generally combined with an incision into the prostate gland. This was a method largely employed by Reginald Harrison, commencing in 1881, his first

operation having been performed on the 4th of November of that year. He used a small perineal incision, opening the membranous urethra; then the prostate was incised, and a perineal tube was introduced and retained for from six to twelve weeks. If the natural channel was not restored the fistula persisted. Prof. Gouley, of New York, claimed priority over Harrison in the reintroduction of perineal prostatotomy, his first operation, in which, however, he left no instrument in the bladder, having been performed April 27, 1880, and his third operation, in which he left a large-sized rubber tube in the perineal wound, having been done in January, 1881. Whitehead was likewise among the earlier advocates of the treatment by a permanent perineal opening.

Excision of the obstructing parts of the enlarged prostate by suprapubic cystotomy was first widely advocated by McGill, of Leeds, in 1887, he having before that date practised permanent suprapubic drainage, which he preferred to that by the perineum. Belfield, in America, had done suprapubic prostatectomy before that time, his first operation being in October, 1886; Dittell had in 1885 removed a portion of an obstructing prostate through a previously existing suprapubic fistula, which he enlarged for the purpose; Trendelenburg, in May, 1886, and Benno Schmidt, in August of the same year, had employed this route for removal of pieces of the prostate; but to McGill has always rightly been attributed the priority in bringing this procedure prominently before the profession. The most enthusiastic supporters of McGill's operation have been Buckstone Brown, Kümmel, Atkinson, Keyes, and Fuller. As originally practised this operation consisted in cutting off, through the usual incision for suprapubic cystotomy, by means of scissors or rongeur forceps, twisting off with bladder forceps, strangulating with an écraseur, or crushing with a lithotrite, any projecting masses of prostate tissue. At times, also, portions of tissue forming the so-called prostatic tumors were enucleated with the finger, either alone or aided by the scissors or other instrument, from their position deep within the gland.

But to Freyer is undoubtedly due the credit of bringing before the medical world a plan of operation whereby an attempt is made to enucleate the whole of the enlarged prostate gland. Through a

suprapubic incision he opens the bladder, and incises the mucous membrane overlying the affected gland, by a cut parallel to the urethra, over each lateral lobe of the prostate. Then by working merely with the finger he states that he has been able to enucleate the entire prostate, leaving the urethra intact. The technique of this operation will be discussed in greater detail on a subsequent page. Many other surgeons have assailed Mr. Freyer's claim to originality, and assert that he is laboring under a grave misapprehension if he thinks he is the first person to operate in this manner, insisting that his method is nothing more than the removal of very large prostatic tumors from the substance of the gland, leaving behind the outer margin of glandular substance which by the growth of these tumors has been compressed into a thin capsule-like layer. Thus, Wallace says: "The more rapidly growing areas (of the diseased prostate) increase at the expense of the more slowly growing ones, which are compressed and stretched over the surface of their quickly growing neighbors. By this process a capsule is formed, ill-defined at first, but later becoming very distinct. The elements forming this capsule show in process of time a lamellar disposition. The adenomatous mass can now be easily enucleated, and not only presents a smooth surface but also leaves behind a smooth cavity." One prostate which he describes, removed post-mortem, showed within the layers of the "capsule" a small lenticular focus of glandular tissue. He concludes: "These facts seem to leave no reasonable doubt that the so-called total prostatectomy is nothing more than the removal of adenomatous masses."

It seems a pity that so many controversies in regard to surgical priority are so constantly arising, and it appears that prostatic surgery is particularly unfortunate in this respect. Riolanus bitterly denounces his contemporaries for claiming as their own operations which had been employed before their grandfathers were born and for a hundred years before that time. Mercier asserted that Leroy d'Etiolles has assumed not only the credit of operations which were not his own, but had also assumed a name not his own, since in reality Leroy came from Paris and not from Etiolles. Gouley spoke almost venomously against Mr. Harrison; and I think Mr. Freyer would be well able to reply to his critics as Mr. Harrison did to Professor

Gouley: "I see that Dr. Gouley claims priority for the proceeding just described; what is of more importance is that it has received his approval."

Prostatectomy by the perineal route, developing from perineal prostatotomy, has been supported by Dittel, Annandale, Harrison, Zückerkandl, Watson, Syms, Ferguson, Moore, Murphy, Bryson, and others.

The simple perineal operation is done through a straight median incision. To gain more room this has been supplemented by an oblique cut on each side, as advocated by Murphy; while Zückerkandl advises a transverse semicircular incision, with its concavity toward the rectum, which is separated from the anterior structures by blunt dissections; and Dittel aims to get still more room by an incision completely encircling the right side of the anus from the coccyx, and continued forward in the median line of the perineum; by this approach he is enabled to remove a wedge-shaped piece of each lateral lobe. The coccyx may be excised if necessary for completing the operation.[1]

Combined operations by the perineal and suprapubic routes have been advocated by Nicoll, who pushed the prostate into the perineum (whence it is removed) by the fingers of one hand introduced into the bladder through the suprapubic wound, and are employed by a number of operators at the present day, including Alexander and Guitéras, some of them removing the prostate through the perineum, and some through the suprapubic wound, using the counteropening to admit the fingers of the other hand to assist in the manipulations.

A mode of treatment by castration introduced in 1893 by J. William White, though widely employed by some surgeons for several years, is no longer in favor. Vasectomy, suggested as a less severe and mutilating operation by Mears, seems likely to continue in use for certain cases for a long time. The mortality from castration for enlarged prostate is at least 18 per cent., taking all cases together; and in selected cases has been reduced only to about 8 per cent. Castration for enlarged prostate was first performed

[1] Perineal prostatectomy for malignant growths was employed in 1866 by Küchler, about 1870 by Billroth, in 1873 by Demarquay, in 1876 by Langenbeck, in 1882 by Stanton, and in 1883 by Leisrink. See Leisrink, Archiv f. klin. Chir., 1883, xxviii., s. 578.

by Ramm, of Christiania, and has been advocated by Griffith and Mansell Moullin.

Ligation of both internal iliac arteries, to induce ischæmic atrophy of the prostate, was proposed by Bier, and employed by him in three cases, one of the patients, operated on intraperitoneally, dying from septic peritonitis. Willy Meyer employed this operation in two cases, the first patient recovering, after secondary hemorrhage and partial gangrene of the left foot; but the second died, apparently of renal disease. Those patients who survived (three out of five) are said to have had their bladder troubles more or less relieved.

Anatomy. In the human fœtus the prostate gland is a paired organ consisting of two lateral masses which coalesce around the first part of the urethra about the fourth or fifth month of intra-uterine life. In some of the lower animals these lateral portions remain distinct and never unite in the median line. These facts have been known for centuries, and it was not until about one hundred years ago that Sir Everard Home took it upon himself to name a "third lobe," that portion of the prostate lying above the ejaculatory ducts and behind the urethra. His experiences were apparently confined to an examination of four or five cases, the majority of which did not show any distinct third lobe. It is further worthy of observation that almost every author since his time has spoken of the "so-called third or median lobe" of the prostate, as if there were reasonable doubt whether such a thing really existed.

Projecting into the prostate gland from the urethra is found the "uterus masculinus," which is the homologue of the female womb, being the persistent lower extremity of the Müllerian duct. This structure has a muscular and glandular wall of its own, and though in such close relation with the prostate has no physiological connection with it; that is to say, the prostate gland, though composed largely of unstriped muscular tissue, can in no respect be considered homologous with the uterus.

Microscopically the prostate is found to be a compound tubular gland, its numerous acini being supported in a framework of connective tissue and involuntary muscle. The involuntary muscle is arranged in a rather compact layer around the periphery, and also

at the site of coalescence of the two lateral lobes above and below the urethra. The unstriped muscular fibres of the bladder wall are prolonged around the prostatic urethra as an inner longitudinal and outer circular layer.

The whole gland is enveloped by a fibrous capsule distinct from the rectovesical layer of the pelvic fascia, and formed by a condensation of the stroma of the gland. Outside of this fibrous capsule, and between it and the pelvic fascia, is found the prostatic plexus of veins, together with the arteries, lymphatics and nerves distributed to the glandular structure. The venous plexus lies chiefly on the anterior and front lateral portions of the gland, receiving the dorsal veins of the penis.

Above, the prostate is in intimate relation with the urinary bladder, lying immediately beneath its neck, and being separated from its muscular walls only by a layer of fascia; below and posteriorly it impinges upon the rectum, connected rather firmly to this structure by a prolongation of the retrovesical fascia. In some instances the prostate gland has been found to envelope only about three-fourths of the calibre of the urethra, this canal merely grooving the gland upon its upper and anterior surface; but in the vast majority of cases the urethra appears to be channelled out of the substance of the gland, somewhat anterior to its midportion.

Beside the support it receives from the bladder and rectum, the prostate is suspended in the anterior fibres of the levator ani muscle, called by Santorini the levator prostatæ, and anteriorly is supported by the puboprostatic ligaments, or true ligaments of the bladder (prolongations of the rectovesical fascia); while in the median line anteriorly, where the rectovesical fascia and the deep layer of the triangular ligament unite, the prostate is in relation with this latter structure, a levator prostatæ muscle being on each side.

Coursing through the posterior midseptum, dividing the two lateral lobes, are found the ejaculatory ducts of the seminal vesicles and vasa deferentia. They empty into the floor of the prostatic urethra on each side of the caput gallinaginis and posterior to the orifice of the uterus masculinus. The prostatic acini of each lobe likewise empty into the prostatic urethra by eighteen or twenty ducts on their respective sides of the median line.

Where a projection into the bladder posterior to the vesical orifice of the urethra appears to arise from the prostate it has been said (Griffiths) in some instances to spring from an accessory prostatic gland, which might then well be called a third lobe. But in the majority of cases such a growth may be, and frequently has been, traced to one or other of the two lateral lobes, being connected to them by a pedicle, much as a subperitoneal fibroid is attached to the uterus. It seems, indeed, not impossible that in those cases where it has been said that no such attachment could be found, the growth had finally torn its pedicle loose, and might in time even have migrated, as is not infrequently the case with the analogous tumors of the uterus. Be these facts as they may, it is undoubtedly true that most surgeons at the present day are inclined to regard the third lobe as non-existent, that portion of the gland formerly called the third lobe, and lying between the two lateral lobes, above and posterior to the ejaculatory ducts, being recognized as merely the area of coalescence of these lateral lobes.

Etiology. The causes of enlargement of the prostate are still involved in much obscurity. Our ideas appear to have become less positive in this respect in proportion to the increase of our knowledge in other directions.

In the first place, nevertheless, practically everyone is agreed that there are two main pathological processes by which enlargement of the prostate gland is effected. Griffiths[1] thought that the adenomatous change was merely the first stage, of which fibrous overgrowth formed the final change: one by means of a tumor formation—a fibromyomatous, or fibroadenomatous, or adenomyomatous change—and the second by a general increase in the size of the organ without distinct tumor formation. Both processes may coexist.

The tumors may be several in number, or there may be only one; they may arise in the substance of the gland or beneath its capsule. They may be composed largely of glandular structure, of muscular tissue, or of fibrous tissue; probably a combination of the first two is the most frequent, producing adenomyomata, the second

[1] Journ. of Anat. and Physiol., 1889, vol. xxiv. p. 236.

in frequency of occurrence being the fibroadenomata, for in scarcely any instance is there no adenomatous tissue whatever.

Where general increase in size occurs, approaching more nearly a true hypertrophy, the fibrous is usually the predominant tissue, and it is the type of enlargement which is, as a rule, the cause of the stony hardness observed in some prostates.

To ascertain the underlying causes of the enlargement, whether neoplastic or hypertrophic, affecting the prostate, a number of theories have been proposed. These may all be reduced to two classes, in one of which the enlargement is held to be primary—to be itself a cause to arise *de novo;* and in the other of which it is considered as secondary change—as the result of some other process, as, perhaps, a misdirected compensatory hypertrophy.

To be strictly sincere, it does not appear to me that it is a matter of very great importance under which of these two theories we enlist our intellect; for it is, after all, a matter of purely academic interest, and for practical surgeons will remain such until some happy discovery will reveal the true cause of tumor formation in general or the reason why at a certain age the bodily powers begin to fail and the tissues become sclerotic.

To my mind those prostates which show tumor formation are to be classed with other tumors, which were formerly defined as discontinuous, purposeless hypertrophies of no known cause, while those glands which show only general hypertrophy are classed with other changes incident to age. The discussion of the causes has, I think, been much confused by the neglect of writers to keep these two classes distinct. Guyon and his school, who held that such enlargement was merely a part of a general senile change, appear to me to confine their considerations to the latter class; and here too may be placed the theory of Harrison, that the hypertrophy results from a vain effort to empty a bladder whose base has previously descended into the pelvis below the level of the urethra.[1] While, on the other hand, Velpeau, Paget, and Billroth, with their followers, who maintained that these phenomena were due to a myomatous change strictly analogous to that found in the uterus,

[1] Were this theory correct, a cure might be hoped for, in cases not too far advanced, by a ventrosuspension of the bladder.

evidently fixed their attention exclusively on cases of what I have called the first class, which is, in fact, the larger.

In discussing the clinical causes, also, it is well-nigh impossible to avoid theorizing, since as I have said above, so little is accurately known of the causes of this affection.

Race does not exert a very marked influence; the negro race is probably slightly less predisposed than is the white, and among the natives of India, China, and Japan, prostatic troubles are considered to be extremly rare. In Turkey, however, they are comparatively frequent (Wishard).

Age appears to exert a very marked influence. It is rare for any symptoms to be produced by an enlarged prostate before a patient has reached the age of fifty years: but Dr. Mudd has seen cases in a young negro of twenty-seven years, in a child of five years, and in an infant of thirteen months. The researches of Thompson and others have shown that appreciable enlargement exists in about one-third of persons over sixty years of age, but that it produces manifest symptoms in only about one in every twenty. For prostatic enlargement to appear first after the seventieth year is very unusual. In natives of India and other tropical climates it appears much earlier, as a rule, the span of life being about fifteen to twenty years shorter than ours.

Social habits exert some influence. The gouty, those who have been addicted to the excessive use of alcoholic beverages, or who have been high livers in other respects, are believed to form a large proportion of these patients. Why this should be so, why such a life should produce enlargement of the prostate gland has not been determined. Excessive sexual indulgence has been held responsible in some instances, the overexercise of the gland being thought to lead to hypertrophy, just as Harrison thought hypertrophy ensued from constantly recurring efforts to empty an atonic bladder. This "prostatic overstrain," so named by Lydston, has been explained in the following way by Harrison: "That the withdrawal of a portion of that function of the prostate in which it has been the most vigorously engaged should be followed by a continued activity in which growth is substituted for secretion, is not, I consider, pathologically illogical." McGuire held that although prostatic overstrain might

be the true cause of the enlargement, yet it was the senile sclerotic changes in the bladder which first caused the enlarged prostate to give symptoms.

Preceding inflammations, such as gonorrhœa, acute or chronic prostatitis, or prostatitic abscess, may also be the starting point of chronic enlargement of this organ.

Chronic passive congestions, finally, as in other organs, may lead to prostatic enlargement as well. Old men with cirrhotic kidneys and liver, with hemorrhoids, or with varicose veins in the legs, are very apt to be sufferers from this malady.

Clinical Pathology. As the prostate gland enlarges, whether from tumor formation or as a result of a general hyperplastic process, various changes are produced in the bladder, the urethra and the rectum. The usual direction of greatest enlargement is parallel to the urethra and posteriorly; that is, in the direction of the least resistance, toward the base of the prostate and away from the triangular ligament, this latter attachment of the prostate being practically a fixed point. The bladder cavity is likewise usually encroached upon, the vesical mucous membrane being raised posterior to the orifice of the urethra, and a pouch forming behind this projection. If the two lateral lobes enlarge in nearly equal degree a mere fold of mucous membrane may be raised across the neck of the bladder, just behind the vesical opening of the urethra, but some little distance in front of the urethral openings. This fold constitutes the most usual variety of "bar at the neck of the bladder."

As the lateral lobes enlarge the prostatic urethra becomes lengthened, sometimes measuring as much as four inches, instead of barely an inch, which is the normal, and the curve is also much increased, from the height to which the inner opening of the urethra is raised.

If one lobe enlarges more than the other, the urethra is correspondingly deflected toward the less enlarged side. Where a submucous tumor projects into the floor of the bladder, at or near the median line, the urethral channel may become Y-shaped, and the catheter will pass now to the right, now to the left of the projecting mass. Finally, where the anterior commissure, or isthmus of the gland,

enlarges as well,[1] there may be a collar formed around the vesical orifice of the urethra, which may then appear as a dimple deeply seated near the centre of the prostatic mass.

At times where no intravesical growth at all is found, or after any such has been removed, it has been found still impossible to introduce a catheter through the urethra, the obstruction then being due to irregular projections into the floor of the prostatic urethra. This circumstance has probably been the cause of many failures to restore voluntary micturition after the old operation of McGill.

The calibre of the prostatic urethra sometimes becomes so much increased from the irregular enlargement of the prostate that it may contain an ounce or two of urine, and the unwary may be deceived into thinking the bladder has been reached by a catheter which merely empties this little collection of urine in front of the real obstruction. On the other hand, the whole prostatic urethra may be compressed into a slit-like channel, impervious to fluid, except when distended by an instrument. The vesical orifice may assume a crescent shape, the concavity of the crescent being directed toward the area of greatest enlargement.

If the enlargement proceed chiefly toward the rectum that channel may be obstructed, and much difficulty in defecation ensue, while hemorrhoids may arise, either consecutively or from a common cause.

Ordinarily the first consequence of the almost invariable post-prostatic pouch formation is the presence of residual urine which the patient is unable to expel by his voluntary efforts. Unless the prostatic enlargement is arrested, the amount of residual urine gradually increases, owing both to the increasing obstruction and to the decreasing muscular tone of the bladder, which becomes weakened by straining and overdistention. Ammoniacal decomposition of the urine, with consequent cystitis, quickly supervenes, and gives rise to what is commonly the first symptom, namely, increased frequency of micturition.

As the residual urine accumulates the vesical orifice of the ureters may first become compressed, preventing the periodical discharge of their contents into the bladder, and causing circulatory disturbances

[1] Dr. Murphy denies that this portion of the gland is subject to enlargement. Journ. Am. Med. Asso., March 29, 1902.

in the kidneys; while at a later period they may become dilated, along with the stretching of the rest of the bladder wall, so that they are no longer protected by a valve, but are in direct communication with the bladder, the backward fluid pressure reaching even the renal tubules. Finally, when the bladder becomes overdistended, a condition of urinary overflow may occur, and the patient will be forced to pass a drachm or less of urine every ten or fifteen minutes, or will suffer from a constant dribbling throughout the twenty-four hours. The combined backward pressure and septic changes in the urine produce renal disease, and this in turn reacts on the composition and quantity of urine.

The bladder may not, however, dilate; its walls may become much thickened, corrugated, and pouched; its cavity may even contract, or may be nearly completely filled by an adenomatous prostate. But as a rule where an adenomatous change occurs in the prostate the bladder dilates; whereas if the prostate be a hard fibrous mass, and comparatively small, the vesical walls are contracted and thickened.

The weight of diseased prostates varies from one to twelve ounces; yet the largest in bulk may not weigh as much as one smaller but more compact.

Uric acid or phosphatic calculi frequently form in the puddle of residual urine, and much increase the suffering of the patient. Hemorrhage may be thus accounted for.

Symptoms. The symptoms of prostatic enlargement may be conveniently considered under the heads of those of which the patient complains and those which the surgeon discovers on examination.

The patient will probably for a rather indefinite time have been affected, perhaps without paying much attention to it, with difficulty in starting his stream of urine; the urine when started will have been expelled with little or no force, tending to drop vertically from the meatus, not spurting forth in the normal parabolic curve; a longer time than usual will have been occupied in emptying the bladder, and at the conclusion of the act of micturition there will have been considerable dribbling of urine, no power existing to void the last drops in spurts. The stream will probably not be smaller than in health unless stricture exists as well.

But probably the first deviation from normal which will have forcibly arrested the attention of the patient will have been the necessity of arising once or oftener during the night to evacuate his bladder. This increased frequency of micturition may also have annoyed him during the day, but is less noticeable than at night. The bladder may become so extremely irritable that no sooner will any urine have been discharged into it from the kidneys than an uncontrollable desire to empty the bladder will arise, and thus the patient will be reduced to a most miserable state, being compelled to urinate every ten or fifteen minutes, and his general health in a very short time showing the effects of this constant unrest. On the other hand, the residual urine may gradually accumulate, without causing such severe distress, and a state of retention with overflow finally develop.

Besides the cystitis thus manifested by crebruria, a feeling of weight and distention in the perineum may exist, and piles, with prolapsus ani, from the consequent tenesmus, may ensue.

The patient may consult a physician before this stage is reached, or he may not be disturbed sufficiently to think this necessary until some exposure to cold, some indiscretion in diet, or some other irregularity in his life has brought on an attack of acute retention of urine. Thus when first seen by the physician he may present a bladder distended nearly to the umbilicus, he himself not being aware that residual urine has been accumulating for perhaps months past. Hence, the first physical sign to be looked for in patients suspected of prostatic troubles is a hypogastric tumor with the characteristics of a distended bladder. It is especially important to detect this before introducing a catheter in cases where chronic retention has probably existed for a long time; for, as is well known, the complete sudden excavation of such a bladder is, as a rule, speedily followed by the patient's death.

The patient should next be requested to urinate, when much information may be derived from the character of the stream, in accordance with the changes noted above. If it is apparent that no excessive amount of residual urine exist, it will not be advisable to introduce a catheter, noting the presence of strictures, the distance of the vesical orifice of the urethra from the meatus, and any changes in the curve or deviation from the right line of the sub-

pubic urethra. Then the amount of residual urine is determined, not being deceived by the ounce or more which may, in rare instances, flow from the dilated prostatic urethra. While the catheter or a sound is still in the bladder a finger should be introduced into the rectum, when, by careful manipulation of the one and palpation with the other, a fairly accurate idea can be obtained as to the size, form, and density of the prostate. The condition of the bladder walls, whether rugous and contracted, coated with phosphates, thickened, or dilated and overstretched, can also be ascertained, as well as the existence of a calculus.

If an obstruction to the catheter be encountered within less than seven inches from the meatus it is in all probability due to a stricture, and not to the prostate; but if the obstruction be easily overcome by sharply depressing the handle of the instrument, if of metal, or by slightly withdrawing the stylet, if of webbing, the obstacle is almost certainly an enlarged prostate.

Diagnosis. The symptoms and physical signs being well known, a diagnosis is not, as a rule, difficult. The most important affections with which enlargement of the prostate may be confounded are: atony of the bladder; cystitis, especially chronic; stricture of the urethra; vesical calculus; tumor or tuberculosis of the bladder, and malignant disease of the prostate or some neighboring structure in the pelvis. By symptoms alone no one of these can be definitely excluded, but by careful attention of the clinical history, combined with painstaking physical examination, I believe that in almost every case a correct diagnosis can be reached.

In atony of the bladder arising from other causes the existence of an enlarged prostate may be readily excluded by combined exploration with an intravesical sound and a finger in the rectum; while in uncomplicated cystitis these same marks of difference hold good, and there is, moreover, no residual urine.

In stricture of the urethra the obstruction, as stated above, is almost invariably within seven inches of the urinary meatus, and an enlarged prostate may be excluded by the means of examination already indicated. In case, however, of an impermeable stricture with chronic retention it will not be possible to satisfactorily examine the prostate until these conditions are relieved.

Where a vesical calculus exists it is not liable to be mistaken for an enlarged prostate unless it is both encysted and so thickly coated with mucus that no grating sensation is imparted to the sound. But even under such circumstances there may be no residual urine, which is, as already insisted upon, a nearly invariable accompaniment of every enlarged prostate producing symptoms, and there will probably not be the characteristic change in curve of the subpubic urethra. If the calculus be prostatic, or even if it merely coexist with an enlarged prostate, a positive diagnosis is more difficult. Bleeding is more common in cases of calculus than in those of enlarged prostate alone, and the pain is less constant and more confined at times when the bladder contracts upon the concretion or when the patient is actively moving about. The greatest frequency of urination, also, is during the day, and the patients with stone are not, as a rule, disturbed much at night.

Probably the most difficult diagnosis of all is that from polypoid growths in the bladder, which, when springing from the region of the prostate, may very closely simulate a pedunculated tumor of this organ. But in nearly all other forms of vesical tumor other than prostatic, hemorrhage is an early and conspicuous symptom, and is usually not attended by much pain.

Tubercle of the bladder is probably always secondary to similar disease elsewhere in the body, most often in the epididymis. While the vesical tenesmus may simulate that present in cases of enlarged prostate, physical examination with sound, finger, and cystoscope, and careful examination made for tubercle vesicule, and the tuberculin test applied, will usually reveal the true state of affairs.

Malignant growths of the prostate itself can be distinguished from benign enlargement of this organ only by the tendency they exhibit to infiltration, if of carcinomatous origin, or to rapid growth and the production of cachexia, if of a sarcomatous nature. Compared to the benign overgrowths they are distinctly rare.

Prognosis. A question of considerable importance and much interest in connection with enlargement of the prostate is that of prognosis. In few other diseases is it so necessary for the surgeon to know what may be accomplished by the various methods of treatment possible, and in probably no other class of cases is the surgeon

blamed more severely for error in judgment. It is not sufficient, indeed, it is neither ethical nor human, to hope that the patient will die of some intercurrent affection before any necessity arises for instituting active treatment on behalf of his enlarged prostate, and hence every physician or surgeon who has such cases under his care must give careful thought and attention to each individual patient, and must know whether the expectation of life will be lengthened or decreased by the treatment he proposes undertaking, or whether the certainty of a life of considerable discomfort for a longer time is not less to the patient's ultimate advantage than the risk to life incurred by a rather severe and shocking operation, which, if successful, will enable the patient to live out his natural term of life in ease and comfort.

There are, then, two main questions to be solved in this connection, first, whether the patient's life can be saved, prolonged, or at least not sacrificed, by the treatment to be pursued; and, second, whether his sufferings will be relieved wholly or in part, or whether no change at all can be obtained.

Under medical treatment and catheterization there is practically no possibility of directly terminating the patient's life; his life may be prolonged, and in certain cases made very comfortable. Many a patient who has to pass a catheter only once or twice in the twenty-four hours will live a life of perfect ease and will round out his days without interruption. But, where the catheter has to be passed frequently—that is to say, as often as four to six times in the twenty-four hours, or where its passage at even longer intervals is attended with pain or difficulty, catheterism must be considered at the present day an insufficient remedy, except in those who are already on the threshold of the grave. The expectation of life, moreover, in patients treated by catheterization has been shown by Harrison and Lydston to be in the average no more than four or five years. The next mildest form of treatment is drainage of the bladder. By this means may be obtained relief of the cystitis, and consequently of the tenesmus, pain, and general unrest in a certain number of cases. In my opinion it is applicable chiefly to those in a very debilitated condition or to the very old. Drainage by a permanent catheter introduced through the urethra can rarely long be endured, and is

usually only to be employed in preparing the bladder for radical operation.

Cabot,[1] in an article on the effect of drainage on the kidneys in nephritis, makes some observations of very great interest in connection with the treatment of prostatics. Starting with the proposition that urinary obstruction from enlarged prostate or other cause produces increased renal pressure which may reach even to the Malpighian glomerules, and that this pressure alone, even without infection, will result in fibrous changes (interstitial) in the kidneys, as well as in an increase in quantity and a decrease in specific gravity of the urine excreted, he goes on to show by the report of six cases that the invariable result of relief of the obstruction was to bring the urine back to a more nearly normal state. Especially noticeable was this result in cases where even the greatly increased secretion of urine had been ineffectual in "carrying off the waste products in sufficient amount," and where uræmic symptoms had appeared.

He is well aware, he states, that this procedure is no new therapeutic measure, but he thinks its applicability to renal disease has not hitherto been sufficiently appreciated, although the good effects of Edebohls' operation in relieving renal tension are well known to the profession. Those authors as Langlois, he adds, who have noted the decrease in size of enlarged kidneys succeeding upon bladder drainage have thought it was due to either relief of congestion or the emptying of hydronephritic pouches.

Dr. Cabot says that for the purpose of the present investigation he has "taken it for granted that a patient with obstructive disease of the prostate and dilated bladder may be regarded as having some degree of interstitial nephritis if the urine is abundant and of very low specific gravity. If such a patient is suffering from anorexia, nausea, and dry mouth, either with or without physical disturbances, these are to be looked upon as symptoms of uræmia due to this interstitial nephritis. If, in addition to these symptoms, he has a high fever and the urine contains pus, it is to be regarded as probable that he has pyelonephritis." By instituting continuous drainage,

[1] Boston Medical and Surgical Journal, 1903, ii. 559.

and by flushing out the urinary tract with large draughts of water, he has succeeded in promptly relieving such symptoms.

The time during which this constant drainage must be continued varies with the individual patient; by discontinuing it too soon, the renal and uræmic symptoms were found to promptly recur; but by persisting in this treatment until the urine was nearly normal in both quantity and quality, his patients returned to a life of intermittent catheterization (when they declined operative interference) in the enjoyment of excellent health.

Dr. Cabot further insists that the dangers of sudden emptying of a chronically distended bladder are much mitigated by the constant drainage instituted; for he has found that although considerable hæmaturia might ensue for a few days, yet that this neither in any way interfered with the perfect drainage, nor was it followed by increasing uræmic symptoms, as is usually the case when such a bladder is emptied and no constant drainage instituted.

The first case reported is that of a man, aged sixty-seven years, who, after using the catheter for some months, there being over twelve ounces of residual urine, was taken severely ill, with a temperature of 103° F.—apparently commencing pyelonephritis. By constant drainage the amount of urine was in the course of some weeks reduced from sixty to seventy ounces to less than fifty ounces daily, while its specific gravity increased from 1007 to 1023. Intermittent catheterization was then instituted.

In the second patient there were anorexia, dry mouth, much nausea, and marked somnolence. By constant drainage the return to normal was remarkably rapid.

The third patient, in much the same general state, had fever as well; his bladder also was distended nearly to the umbilicus. Thirty-seven ounces of urine of very low specific gravity were withdrawn, and by continuous drainage in a period of two months the patient's health was practically restored, the quantity of urine, however, remaining at seventy to ninety ounces daily, and its specific gravity being about 1008.

The fourth patient, aged seventy-two years, had suffered for several years with crebruria, there being about twelve ounces of residual urine. A resort to catheterization once or twice daily produced in

about two weeks' time symptoms of commencing pyelonephritis. Constant drainage was then begun. Hæmaturia occurred, and the next day the patient became uræmic. Under proper constitutional treatment all unfavorable symptoms disappeared, but it was not until the seventy-ninth day that intermittent catheterization could be substituted for continuous drainage. His daily urine remained about one hundred ounces, of specific gravity 1012 to 1015.

In the fifth patient the chronically obstructed bladder contained forty ounces of urine. Constant drainage, while it produced transient hæmaturia, relieved the delirium, fever, and thirst. Three weeks later intermittent catheterization was begun. The urine at last report, over a year after the acute attack noted above, was forty to sixty ounces daily in quantity, of specific gravity 1017.

The last patient reported was in the early stages of chronic nephritis, with polyuria amounting to eight pints in the twenty-four hours. When chronic retention set in this amount was reduced to six pints, of specific gravity 1005. Constant drainage was instituted, and in one day the amount of urine drained off reached sixteen pints. This was gradually reduced to six pints, and finally to four pints daily, while the specific gravity rose to 1012 and 1015.

The success of Thompson, McGuire, and others in treating these patients years ago by means of suprapubic permanent drainage, and of Harrison by means of a perineal tube, should not be forgotten at the present day; and while we recognize the inadequacy of such methods to restore the patient to his normal condition, yet in a limited number of cases they are still useful. Especially is this so in patients with very bad cystitis and where some immediate relief is imperative. In such cases so radical an operation as prostatectomy will almost surely kill, unless time can be obtained to relieve the cystitis, to get the kidneys into fair condition, and to improve the general health of the patient. In patients such as these the formation of a permanent suprapubic fistula by McGuire's method, or, if the prostate be not too large, simple perineal prostatotomy, with the introduction of a tube, will afford almost certain relief to the urgent symptoms, and in many instances will enable the constitution to withstand prostatectomy at a later date. Neither castration, vasectomy, nor ligation of the internal iliacs will act sufficiently

quickly in these cases; indeed, in my opinion, these operations are no longer to be considered desirable under any circumstances.

References which I have to 107 cases treated by McGuire's method show only two deaths, so that even in the most serious cases it will be exceedingly unlikely to precipitate a fatal termination.

The Bottini operation is not one for the general surgeon, nor, to my mind, for the average patient. On the surgeon's part it requires a technical knowledge which, even in the hands of those known to be the most skilful in its employment, has not sufficed to prevent fatal complications. The greatest points in favor of galvanocaustic prostatotomy are its low mortality (4 to 6 per cent.), and the fact that if it fails to relieve after the first application it may be repeated once or several times with little more risk than attended its first employment. Moreover, the patient has still a prostate left whose removal by excision may effectually cure him even when several applications of the Bottini cautery have proved ineffectual. On the patient's part this operation is often welcomed for these very reasons: that its danger is comparatively slight; that general anæsthesia is not required; that no "cutting" is to be done; that no external wound is left for healing, and that it does not interfere with subsequent operative treatment by other methods.

But with all the advantages claimed for it, Bottini's operation is first and foremost a makeshift, quite as much as castration or McGuire's suprapubic fistula. It does not remove the offending organ. It burns grooves in it and the surgeon leaves sloughs to be cast off into the bladder, and then waits, hoping that the cicatrization of his sloughing sores will diminish rather than increase the deformity. It is an operation done practically without the aid of either sight or touch; it is nearly impossible to know how long, how wide or how deep are the grooves burned, or whether they are being burned in the prostate or in the bladder wall, and it is absolutely impossible to know what has been accomplished in destruction of prostatic tissue or that the prostate will not again grow into an obstructing position. Moreover, it is an operation wholly inapplicable to emergency cases and to patients with urethras impassable from either strictures or prostatic obstruction. It is therefore, in my opinion,

an operation only to be employed when (1) prostatectomy is inadvisable or is refused, or when (2) the urethra is freely open to instrumentation, there is no cystitis, and little residual urine.

We are reduced, then, to prostatectomy as the operation of first choice in all cases in which it is not contraindicated. When done by the method of Freyer, that is, by suprapubic enucleation, with the finger passing between the capsule and the sheath, the mortality is decidedly lower than by the method of McGill. In twelve cases of the former operation Mr. Moynihan had only one death; and in fourteen cases Mr. Freyer had no more—a death rate of less than 8 per cent.; Barling, however, reports ten cases so treated, with three deaths, making, with six successful operations recorded by McRae and by Stoker, a combined mortality rate of less than 12 per cent. for the forty-two patients, while the statistics of Belfield and of Mansell Moullin show respectively nearly 14 per cent., and over 20 per cent. for McGill's operation. Recent cases of McGill's operation, reported by Armstrong, Horwitz, Fuller, and Thorndike, show a rate of over 21 per cent. among twenty-eight cases. The mortality in seventy-three recent cases (Albarran, Deaver, Ferguson, Horwitz, Murphy, Syms, Verhoogen) of perineal prostatectomy is only a little over 8 per cent., but the results have not as a rule been nearly so good as those achieved by Mr. Freyer's method. Thirty-four out of forty-two patients treated by this latter operation are reported as having their urinary function restored to its natural condition.

Mere figures, therefore, are decidedly in favor of Freyer's operation; but, both from what I have read in the reported cases of others and from my own experience, I am convinced that it is not the best operation in every case, even if it be possible.

Mr. Freyer reports a case in which he attempted his own method, but owing to the density of the gland and its firm adhesions to the rectovesical fascia, he was able to remove only pieces of it. This patient died on the second day after operation of heart failure, and the bladder was found full of clots, the hemorrhage during the operation having been free. Now it does not appear to me to be strictly sincere to argue that Freyer's operation gives a mortality of less than 8 per cent. when we exclude cases in which it was attempted

but was found impossible of performance. As well might we give a certain mortality for the operation of ovariotomy, considering only those cases where the adhesions to the intestines were few and light and where the cyst shelled out easily, yet exclude those cases where a prolonged and delicate dissection was necessary to deliver the cyst, claiming that such cases form a class of themselves. Such a method of calculation would only lead others astray and encourage us in self-deception, a thing Mr. Freyer himself would be very far from countenancing.

Certainly, everything within my own experience convinces me that while Freyer's method of prostatectomy is by far the best operation for all cases to which it is applicable, yet that there is a small and limited number of cases in which it is neither advisable, nor, as Mr. Freyer's own patient above referred to plainly shows, at all possible.

The perineal operation I have employed a number of times, and have found it quite satisfactory in certain cases, but for some years have largely abandoned it for the operation of complete suprapubic extirpation, which I find in my own hands gives better functional results, is attended by a less mortality, and is certainly easier of performance. Of course, we know that mere facility of execution for the surgeon is no argument in favor of one operation, if another, even if much more tedious and intricate, would assure a better result to the patient; but the greater ease in the suprapubic operation consists not alone in mechanical execution, but in economy of time and less distortion of the neighboring parts, both of which are considerations of importance in the class of patients under discussion.

Treatment. The treatment therefore of prostatics—a convenient term adapted by Belfield from the German *Prostatiker* and the French *prostatique*—resolves itself into a choice of remedies, not into any hard-and-fast rules which may not be transgressed. It has well been said that he is either a fool or at best a surgeon of very limited experience who knows of only one method of treatment for a certain class of cases; and while I myself maintain that prostatectomy by the suprapubic method of Freyer is the best treatment, I do so with the distinct reservation that it is not immediately applicable to every case.

Having seen, then, in the preceding pages the relative dangers and

probabilities of ultimate success from the various methods of treatment applicable, it is next requisite to attempt an expression of some general rules which may guide the surgeon in selecting his course of treatment.

A healthy man somewhat under sixty years comes to you with the simple complaint that he finds it necessary to rise once almost every night to pass his water; that this is becoming, he thinks, a more invariable habit; that he has waited, thinking matters might return to their former state without treatment, but that he fears they are growing worse. In such a case as this, after finding the cause of his trouble to be moderate enlarged prostate, and determining the amount of residual urine to be trifling—one or two ounces—I would recommend the passage of a soft rubber catheter, with strict asepsis, every night at bedtime. It is probable that the bladder being thus emptied, no call during the night would arouse him, and it is barely possible, though I admit very improbable, that with good hygienic surroundings, and a regular life, the bladder might acquire sufficient tone to expel the residual urine, and that no further trouble would ensue. More likely it is that catheterization, at first employed only once in the twenty-four hours, would soon, say in the course of six months to a year, be resorted to twice or even three times in a similar period, to obtain relief from the vesical tenesmus, and that from this constant use, cystitis, if only of a mild degree, would have developed. At this period of the disease it is important to undertake more active measures before the general health is impaired. In cases such as this very many surgeons would be disposed to recommend the Bottini operation—a trifling risk; no cutting, no external wounds to heal, a prostate not very large, nor causing much obstruction; possibly no impairment of the generative functions as a result of the operation. The patient would be more inclined to accept such a proposition than one of radical extirpation of the gland, even though he were told that one operation by Bottini's method was not a sure cure, and that even after two such operations the prostate might still continue to enlarge. In my opinion, if the prostate gave evidence of rather rapid increase in size, showing it to be probably an adenomatous gland, a Bottini operation would not be for the best ultimate good of the patient; I

would advise him to have a suprapubic prostatectomy done by Mr. Freyer's method. The prostate would probably continue to enlarge even if temporary relief from the residual urine had been obtained by the galvanocaustic operation; and the patient would in time be forced to submit to a radical operation when periprostatitis had rendered complete extirpation impossible, when his bladder had become weakened, his kidneys diseased, and his general constitution impaired. If, on the other hand, the prostate in such a case appeared to be a small fibrous mass, not increasing in size, but obstructing the urine by pressure on the prostatic urethra, I should feel less disinclined to try the effects of a Bottini operation. This latter class of patients is distinctly rarer than the former. In the former case the prostate could almost certainly be removed in its capsule, from within the sheath of the gland, with comparatively little hemorrhage and with practical certainty of no recurrence. In the latter case, however, the prostate would probably be densely adherent to its sheath; an attempt at extirpation by the suprapubic route would probably be difficult, bloody, and prolonged; the gland would have to be removed in pieces, not insuring its total ablation, but perhaps producing serious and lasting injury to the vesical sphincter. A perineal operation, removing the floor of the prostatic urethra, and allowing the isthmus of the gland above the urethra to remain, would here I think be attended by better results than a suprapubic operation.

Suppose, now, a second case, similar to that just discussed, but in an older man, from sixty-five to seventy years of age. What treatment is to be pursued here? If his constitutional condition be as good as was that of the patient just mentioned, I would employ a similar treatment and advise suprapubic prostatectomy under the same conditions, but at an earlier date. If postponed too long the health of a patient at such an age may rapidly fail, and any operation prove too late or too great a risk to save his life.

In a third case the patient may first come to us after he has been using the catheter for months or years. He may apply because of the greater frequency with which the catheter must be used, because of the greater pain and difficulty attending its introduction, or for both reasons. In any circumstances he probably has severe cystitis

and impaired kidneys. If drainage by a permanent catheter through the urethra, combined with the internal administration of urinary antiseptics, washing out of the bladder, and careful diet, effects an improvement in these particulars, I would not hesitate to resort to Freyer's operation as the surest method of saving his life and restoring his urinary function, providing the prostate were adenomatous. If no improvement in the cystitis was apparent, I would at any rate open the bladder either suprapubically or through the perineum, and direct my further treatment in accordance with what I then discovered by sight and touch. If the bladder before operation appeared to be small and contracted, and the prostate hard and not very large, I should open the perineum, and at least drain the bladder, and at the same time remove so much of the offending organ as the condition of the patient and the state of the parts appeared to countenance. If the bladder appeared to be dilated and the glands adenomatous, I should prefer the suprapubic route, completing the operation by Mr. Freyer's method if possible, but at least draining.

If drainage only had been instituted, three terminations to the case present themselves: (1) The patient dies from progressive asthenia and nephritis, in spite of the drainage, or (2) he recovers sufficiently satisfied with a permanent urinary fistula to seek no further treatment, or finally (3) he still requires a prostatectomy, which now, in the absence of cystitis, can be performed with little danger.

Still another case may occur: A patient, old, feeble, perhaps delirious, with chronic retention of urine, the urethra not being permeable to instruments. Hemorrhage from the urethra, and perhaps into the bladder, complicates the case. Evidently the patient is in a critical condition. Strictures may make matters worse. At all events, the existence of an enlarged prostate is definitely determined; but the history throws little light on the primary cause of the urinary retention. What is to be done? Castration, ligation of the internal iliacs, the Bottini operation, suprapubic or perineal prostatectomy are all discarded, some as futile, some as preposterous, others again as entailing far too great shock. The only thing left to do is to drain the bladder. I do not think simple aspiration in such

a case as this will be of any lasting benefit, but rather that it will do harm by relieving the tension and encouraging vesical hemorrhage, which in turn only makes matters worse. To relieve the immediate distress of the patient it may be advisable to thus draw off his urine, but it is idle to hope that the obstructions in the urethra will subside soon enough for permanent drainage to be available by that channel, and in my opinion the surgeon had best proceed at once to a suprapubic cystotomy. Such an operation can, if necessary, be done under local anæsthesia from cocaine, and does not involve an incision into the prostate, which would be necessary in a perineal cystotomy, and which would much increase the risk of hemorrhage. The bladder being opened, any vesical oozing could be controlled with hot solution. If the patient did not react from the serious condition in which he was at entrance, his death would at any rate have been rendered easier, and he would have been afforded the only chance to recuperate which surgery has to offer. As in the former case considered, a recovery from the cystitis with persisting debility from the enlarged prostate would be an indication for prostatectomy; or if such an operation appeared to be too severe, a Bottini operation, which could be done through the urinary fistula, if preferred, might be undertaken, with a fair probability of relief for the few remaining years of life.

Technique of Special Operations. *Perineal Prostatectomy.* The patient being placed in the lithotomy position, a medium-sized steel sound—one that will pass readily through the urethra—or a Ferguson's prostatic depressor, is introduced into the bladder, and a straight median incision is made in the perineum from the base of the scrotum to a point near the margin of the anus, of course not dividing the sphincter. The membranous urethra is then exposed in the usual way, and, if preferred, may be opened. The dissection is then continued, dividing the anterior fibres of the levator ani, which are drawn to each side with hooked retractors, and the sheath of the prostate thus exposed. The rectum and its overlying tissues should be pressed backward with a blunt retractor, opening up a fairly large space in the recto-urethral triangle. By the sound or Ferguson's depressor, inverted, or with a finger passed into the bladder through an opening in the membranous urethra, the pros-

tate can now be pressed downward and outward, stretching the process of rectovesical fascia composing its sheath. When this is incised the separation of the prostate from the surrounding structures should be begun at its posterior part, and the operator should work forward. In this dissection, which is by no means as easy as the shelling out of the gland in the suprapubic operation, I have been greatly aided by the use of Murphy's hooks, a full set of which I now regard as indispensable to the proper performance of this operation. By them each lobe of the gland is in turn drawn toward the operator and separated from the bladder. The floor of the prostatic urethra may always be removed, and the upper or anterior commissure of the gland may well be left behind, since in the class of cases in which I recommend this operation there is little likelihood of its causing any subsequent trouble. Nor will there usually be found any pedunculated outgrowth into the floor of the bladder in patients whose hard and fibrous prostates suit them for this operation.

When as much of the gland as seems advisable has been removed and it is evident that the vesical orifice of the urethra is as low as the lowest part of the bladder, a fair-sized rubber tube is passed into the bladder through the perineum for drainage, being stitched to the skin, and is gently packed around with iodoform gauze. If the oozing of blood be considerable, and cannot be controlled by douching with hot solutions, this packing may be quite firmly applied and remain in place for three or four days. A light gauze dressing held in its place by absorbent cotton and a T-bandage completes the operation.

On being returned to bed the perineal drainage is attached by another tube to a bottle hanging beside the bed: The outer dressings may be changed once or oftener daily as required; the packing should be removed about the fourth day, and the perineal tube at the end of a week. Commencing in the second week, a full-sized catheter or sound should be passed through the urethra every third or fourth day. It will commonly be found that before the end of the second week the patient will pass more urine by the urethra than through the perineal wound. He should be kept quietly in bed until the urine ceases to pass by the perineum. To encourage the voluntary passage of urine he may turn on his side or even to the prone position early in the second week.

SUPRAPUBIC PROSTATECTOMY. My own technique for this operation does not differ materially from that described by Mr. Freyer.

The patient being in the Trendelenburg position, and having not more than four ounces of fluid in his bladder, this organ is opened by a suprapubic incision of about four inches in length. The margins of the vesical wound are then to be secured to the abdominal wall by a suture of silkworm-gut on each side. These sutures should pass through the whole thickness of the abdominal wall, and may be either tied and left at the conclusion of the operation or caught in hæmostatic forceps and removed before dressing the wound. The former plan, which makes a ventrosuspension of the bladder, is not without its subsequent advantages.

By thus securing the bladder to the abdominal wall before the enucleation of the prostate is begun we both steady this latter organ and render urinary extravasation between the layers of the abdomen exceedingly unlikely.

A rubber catheter is then introduced into the bladder through the urethra, and an incision of an inch or less in length parallel to the urethra is made in the mucous membrane over the most prominent portion of the prostate as it projects into the bladder. A pair of scissors is safer than a knife for this purpose. The index finger of the surgeon is then introduced into this cut, and readily finds a natural plane of cleavage of the prostate from its sheath. The index and middle finger of the other hand, gloved, are introduced into the rectum, and with the thumb making counterpressure as well from the perineum, the prostate can be enucleated in a surprisingly short space of time, and with unexpected ease. The vesical orifice of the urethra has in my operations generally torn lose from the bladder wall, and the prostate has come away from around it as a glove is stripped off the finger, leaving the proximal inch of the urethra, formerly embedded in prostatic tissue, lying free in the bladder and only attached where it pierces the posterior layer of the triangular ligament to become the membranous urethra. I have removed the entire prostatic urethra in some of the cases from which there has been no untoward results; in fact, I believe that it is the rule for the prostatic urethra to be removed with the prostate. The lobe first attacked should be separated from its attachments, first, poste-

riorly, then on the other side and below, and finally from the triangular ligament at its apex. If the gland be not too large both lobes can be freed and delivered through one incision; but if it be of unusual size it will be found advisable to repeat this procedure for the second lobe through another incision, whereupon each lobe may be delivered separately into the bladder. During these manipulations the original incision in the mucous membrane is of course torn larger by the finger.

By clinging close to the capsule of the gland, or even by passing between its outermost planes of tissue (as is claimed by some, is always done in this operation), the prostatic plexus of veins is not injured, and there is comparatively little hemorrhage. If the veins are laid bare it is evident that the whole capsule has been removed; but if a smooth-walled cavity is left behind, the surgeon's finger has in all probability split the prostatic capsule and leaves a layer of this tissue in place. It makes no difference to the success of the operation which has occurred, except that in the former instance some of the veins might be ruptured and cause troublesome hemorrhage. Especially apt is this to be the case where the prostate is the seat of a fibrous rather than a glandular enlargement, or where periprostatitis has existed.

When the surgeon has removed the prostate he will be very much surprised, as well as pleased, to see with what rapidity the cavity, from which it has been enucleated, contracts. This occurs largely from the pressure of the neighboring structures and from the natural elasticity of the parts, and is an event which diminishes the bleeding. Free douching of the bladder with hot saline solution, which both cleanses and checks the unavoidable oozing, together with the introduction of a suprapubic tube, completes the operation, which usually, from beginning to end, lasts not more than thirty minutes. The suprapubic wound may be closed up to the point of exit of the rubber-drainage tube, the tube being stitched to the skin. It is the practice of the writer to leave the wound open and dress with plenty of gauze and absorbent cotton. A catheter is not retained in the urethra, nor do I drain through the perineum. It is sufficient to pass a rubber catheter, by the urethra, once in every twenty-four hours after the first day or two, and to douche the bladder through it,

allowing the fluid to drain by the suprapubic wound; recently, however, it is not my practice to wash the bladder out, except through the suprapubic wound. After forty-eight hours the suprapubic tube may be removed, and the patient encouraged to pass his own urine. Voluntary micturition commonly returns earlier after this operation than after that by the perineal route; the patient may be propped up in bed on the fourth or fifth day, and be allowed to sit in a chair at the end of a week or ten days, if his general health permits.

In both these operations the power of procreation is, of course, destroyed by the unavoidable injury to the ejaculatory ducts; but, as it is questionable whether the spermatic fluid would be ejected without the contraction of the prostate, and as it is probable that it would not be fertile without the secretion of the prostate, the destruction of these ducts is a secondary consideration. The power of intercourse is less apt to be impaired after a suprapubic enucleation than after a perineal prostatectomy, and in some patients in whom it has been lost prior to operation, this procedure appears to have restored it.

A brief report of the cases, thirteen in number, in which I have had to resort to Freyer's operation, is appended. I wish to take this opportunity of crediting my friend, Dr. Astley P. C. Ashhurst, with much of the work done upon this paper, and in particular the bibliography.

All of these patients were under my care in the German Hospital:

T. F. aged sixty-three years, admitted July 30, 1903; discharged September 7, 1903. Recovered. Present troubles began in May, 1902. Patient had pain upon urination, had trouble in starting the stream, then a sudden stoppage of the stream. At that time he passed two small stones, some blood, but no blood lately. Passes urine quite often, and at night some eight or ten times. Patient says that when he is up and about he has a feeling that there is a foreign body in the bladder.

July 31*st*. A stone was detected by passing a sound into the bladder.

August 3*d*. Operation. Three stones removed from the bladder, diameter of a penny, and about three-eighths of an inch in thickness. Prostate enucleated.

L. F. (No. 1533), aged sixty-eight years. Admitted June 30, 1903, discharged September 11, 1903. Recovered. No venereal history. Bowels are regular. Moderate user of tobacco; no alcohol. One week ago infection of middle finger of left hand. For the past two years patient has had

trouble in passing his urine, often obliged to get up eight or ten times at night. Some burning upon urination, and difficulty in starting the flow. Four days ago inability to pass urine; catheter passed with great difficulty. On admission 600 c.c. of bloody urine withdrawn; a false passage was detected in passing the catheter. Rectal examination shows an enlarged prostate quite firm to the touch.

Operation, July 6, 1903. Suprapubic prostatectomy.

T. C. (No. 1623), aged seventy-seven years. Admitted September 19, 1903. Patient has been suffering from frequency of urination for years, more marked at night. Ten days ago urination became difficult; three days before admission impossible. He was catheterized by physician for two days with difficulty. Third day catheterization was impossible. On admission, bladder greatly distended, reaching to the umbilicus. Prostatic catheter passed; several strictures encountered anteriorly; at the prostatic urethra is a large false passage leading to the left. Prostate greatly hypertrophied, size of small orange. Urine obtained by catheterization very bloody.

September 21st. English catheter passed, permanently fixed in bladder.

Operation, September 23, 1903. Suprapubic prostatectomy. Patient never rallied after operation.

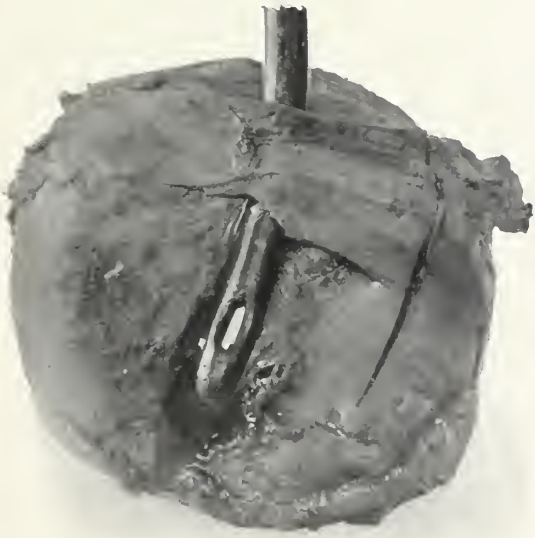
J. M. C. (No. 1555), aged sixty-three years. Admitted July 18, 1903. Discharged August 14, 1903; recovered. Patient uses alcohol moderately, tobacco to excess. Six months before admission patient first noticed difficulty in starting the stream, especially in the morning; as a rule, was compelled to urinate once during the night; during the day passed urine about four or five times. Amount said to be scanty; slight pain on starting. One week before admission was exposed to cold and rain and suffered retention; was relieved by catheter, and since then has been catheterized twice a day. Rectal examination reveals a firm mass at the neck of the bladder about the size of a large hen's egg. Amount of residual urine, 50 c.c.

Operation. Suprapubic prostatectomy.

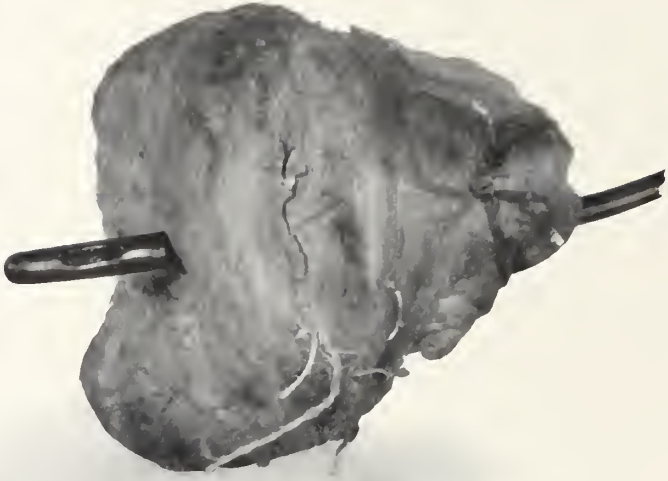
D. D. (No. 1469), aged fifty-eight years. Admitted May 4, 1903. Discharged June 3, 1903. Bowels are regular. Patient uses alcohol and tobacco moderately; complains of burning sensation after urination, about one month before admission had to urinate every ten minutes, passing 10 to 15 c.c. at a time; the stream would suddenly stop and then start up again. Urine highly colored, red, supposed to be bloody. Pain more marked on moving about. Used to have to urinate every twenty minutes during the night, of late has not urinated so often, three or four times. Pain starts just above the symphysis pubis and shoots down the penis, stinging sensation at end of penis.

Operation. Suprapubic prostatectomy.

S. L. T. (No. 1542), aged seventy-three years. Admitted July 9, 1903. Patient has never used alcohol. Had an attack of gonorrhœa when about



CASE III. T. C., aged seventy-seven years. Reduced one-fifth.



CASE IV. J. M. C., aged sixty-three years. Reduced one-fifth.

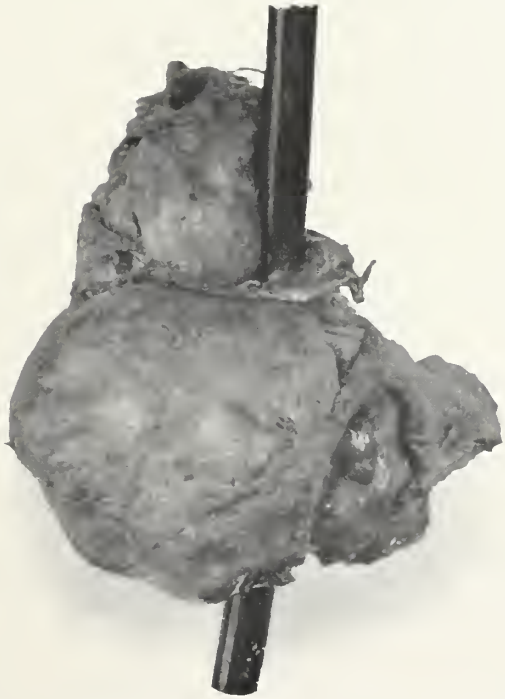
CASE V. D. D., aged fifty-eight years. Reduced one-fifth.

Case VI. S. L. T., aged seventy-three years. Reduced one-fifth.

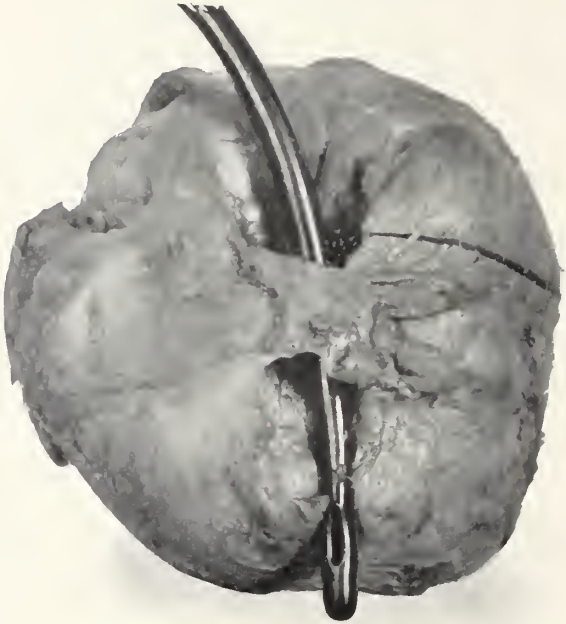
CASE VI. S. L. T., aged seventy-three years. Upper surface. Reduced one-quarter.

CASE VI. S. L. T., aged seventy-three years. Under surface. Reduced one-quarter.

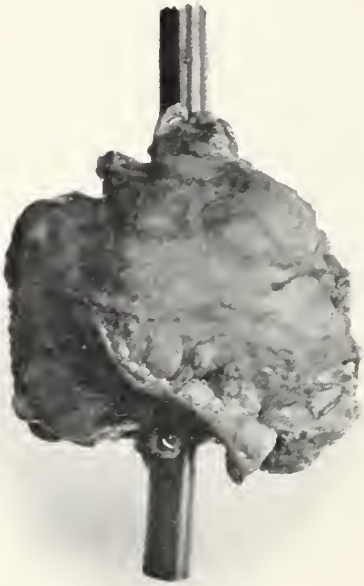
Case XII. A. S., aged sixty-eight years. Under surface. Reduced one-fifth.



Case XII. A. S., aged fifty-eight years. Upper surface. Reduced one-fifth.



Case VII. H. M. T., aged sixty-six years. Reduced one-quarter.



Case XIII. P. J., aged sixty-five years.

eighteen years of age. Present illness began about two and a half years before admission, with frequency of urination, especially at night, obliged to get up about every fifteen or twenty minutes to urinate; often when upon his feet he would involuntarily urinate. Flow lacked force, coming in a thin stream; of late bright blood was present. At the beginning of the illness much sediment was present in the urine. Rectal examination shows an enlarged prostate, very firm, size of a lemon.

Operation, July 11, 1903. Suprapubic prostatectomy. Patient died July 26, 1903.

H. M. Y. (No. 1502), aged sixty-six years. Admitted June 8, 1903, Discharged August 1, 1903. The patient's father died of prostatic disease; a moderate user of alcohol. For past fifteen years had had frequency of urination, most marked at night. Two years before admission suffered from an acute attack of cystitis. In July, 1902, was operated upon for vesical calculus, since which time he has had a suprapubic fistula. Has not passed urine through urethra for six months. Rectal examination shows very hard prostate, size of lemon.

Operation, June 16, 1903. A stone the size of a lima-bean extracted from the bladder. Prostate enucleated.

D. R. Admitted December 9, 1901. For the last eight years patient has been troubled with dribbling urine; for two years had had overflow of retention, due to an hypertrophied prostate. Rectal examination shows prostate size of a small orange, bilateral enlargement.

Operation. Suprapubic prostatectomy. Patient died of uræmia December 23, 1903.

J. S. Admitted February 24, 1901. Discharged April 5, 1901. Seven years before admission patient began to have frequent and painful urination; pain would shoot to penis and groin. Was operated two years later for stone in the bladder; both testicles became involved and were removed. Had had a perineal section done, in which part of the prostate gland was removed one year before the stone was removed. Gradual atrophy of penis. Ever since stone was crushed he had had cystitis. Urinated eight to ten times during the night, and every hour in the day. Prostate hard, not enlarged.

Operation. Suprapubic prostatectomy.

J. J. M., aged sixty-nine years. Admitted September 14, 1903 Discharged October 15, 1903. Six years before admission patient began to have "bladder trouble," frequent urination; for two years had used a catheter; now every two hours, with much pain. Urine very cloudy and ammoniacal odor. By rectal examination a large, soft prostate can be felt.

Operation. Suprapubic prostatectomy.

J. S., aged fifty-five years. Admitted March 19, 1903. Discharged May 9, 1903. Patient uses alcohol moderately, had chewed and smoked to excess until within ten years before admission. No venereal history.

Bowels regular. Four years before admission patient noticed trouble in micturition, slowness in starting the stream, frequency of urination at night; no pain. Used catheter three to five times daily for three years, has no difficulty in passing catheter, and when introduced expulsion from the bladder seems unimpaired. Rectal examination reveals an enlarged prostate, moderately firm, extending posteriorly into the bladder.

Operation. Suprapubic prostatectomy.

A. S., aged sixty-eight years. Admitted March 25, 1903. Patient always enjoyed good health, lived a very active life.

Present history. Had frequency of urination fourteen months prior to admission to hospital, during which time he had used a catheter as often as every fifteen minutes. Residual urine not noted. Has been confined to bed for the last three months with continuous catheterization.

March 26th. Operation. Suprapubic prostatectomy. Suprapubic drainage with half-inch rubber tube. Urine from bladder, through drainage tube, clear.

29th. Tube removed. Drainage from suprapubic wound without tube.

April 6th. Voided small amount of urine by urethra. Patient still under treatment.

P. J., aged sixty-five years. Admitted March 5, 1904. Patient has had frequent urination day and night for some time. Four weeks before admission patient was unable to pass urine; since then he has had to be catheterized, twice a day at first, lately three times a day. By rectal examination prostate is found enlarged. Residual urine, 300 c.c.

March 12th. Meatotomy.

20th. Suprapubic prostatectomy, suprapubic drainage by rubber tube.

23d Urine by tube clear.

24th. Tube taken out.

25th. Small amount of urine passed through urethra. Patient still under treatment.

Statistics.

Suprapubic permanent drainage.

Operator.	Cases.	Deaths.	
McGuire	39	2	
Poncet	35	0	
Horwitz	33	0	
	107	2	Mortality 1.87%

Bottini operation.

Operator.	Cases.	Deaths.	
Bangs	42	3	
Horwitz	33	0	
Meyer	59	7	
Young	41	3	
	175	13	Mortality 7.5 %

(Freudenberg, in 1900, collected 753 operations by Bottini's method, 44 of which, or 5.8 % terminated fatally).

Perineal prostatectomy.

Operator.	Cases.	Deaths.	
Albarran	35	1	
Deaver	5	2	
Ferguson	6	0	
Horwitz	7	3	
Murphy	8	0	
Syms	9	0	
Verhoogen	3	0	
	73	6	Mortality 8.2 %

Freyer's operation.

Operator.	Cases	Deaths.	
Barling	10	3	
Freyer	14	1	
McRea	3	0	
Moynihan	12	1	
Stoker	3	0	Mortality 11.9%
	42	5	

McGill's operation.

Operator.	Cases.	Deaths.	
Armstrong	9	4	
Fuller	5	0	
Horwitz	5	1	
Thorndike	9	1	
	28	6	Mortality 21.4 %

(Belfield, in 1890, collected 88 cases of suprapubic prostatectomy with 12 deaths, a mortality of 13.6 %; and Moullin, in 1892, collected 94 cases with 19 deaths, or 20.2 % mortality.)

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DISCUSSION.

Dr. Thomas R. Neilson: The hour is rather late to enter into a detailed discussion of so interesting, important, and comprehensive a paper as Dr. Deaver's, but I do think that something should be said by the surgeons present. It would be impossible to discuss now the various features of the subject, and Dr. Deaver has gone over the ground so very thoroughly that there remains little for us to speak upon. My own experience in the treatment of prostatics has been peculiar in that I have had almost a succession of wrecks to deal with. I have had considerable experience in observing the effects of the continuous use of the catheter, and I have had some experience in suprapubic removals. Between these extremes I have little to say. I will say this, though, that the operation which has for its object the definite and complete removal of the cause of the trouble is that which should most strongly appeal to the surgeon as well as to the patient. The Bottini operation can never remove the primary *cause* of the obstruction. It may remove that part of the obstruction which annoys and aggravates for the time being, but it does not so correct it that it may not recur. Complete removal of the hypertrophied gland is the only thing that leads to recovery.

Dr. Deaver has given the mortality of the different surgical procedures for enlarged prostate. It is not for me to say anything on that. My own inclination, like Dr. Deaver's, is toward removal by the suprapubic method. I think that by it there is less likelihood of disturbance of other structures. If the prostate is in a condition of adenomatous enlargement the probability of an easy removal is very great. A perineal prostatectomy, on the other hand, may be very difficult. The gland may be far from the surface, requiring a deep wound and considerable disturbance of the parts, and there is the chance of that most annoying sequel, a fistula.

As Dr. Deaver aptly says, the methods chosen must be suited to the particular case in question.

Chronic urinary sepsis is a condition to be reckoned with in a large number of prostatics, as is arteriosclerosis, damaged kidneys, etc. In cases in which the constitutional changes are marked, simple drainage by catheter, or by suprapubic or perineal incision in suitable cases, will give relief.

Dr. Orville Horwitz: The danger to the patient commences as soon as it becomes necessary to resort to daily catheterism. A radical operation performed as soon as symptoms of obstruction begin to appear, with the low mortality which attends such cases, offers the individual a better chance for health, comfort, and prolongation of life than the unfortunate patient who leads a catheter life. Persons who have to depend on constant catheterism eventually succumb to one or other of the sequelæ attendant on this method of treatment. We tabulated in 1892, 886 Bottini operations by 48 operators.

Of these, 84.3 per cent. were cured or improved, 10 per cent. unimproved; mortality 5.7 per cent.

Little need be said in defence of an operation which affords so much benefit with so little risk and is applicable to such a large number of cases. The genito-urinary surgeon who desires to maintain his position in the front rank of the profession can no longer refuse to acquire the skill which is necessary in order to perform it properly. A large number of individuals suffering from urinary obstruction due to this cause are amenable to treatment by means of the Bottini operation. It is the proper method to employ in suitable cases, giving the best results with the least danger. Our opinion is based on an experience of ninety-five operations.

Prostatectomy is as much the subject of controversy as is the Bottini operation. The surgeon who desires to acquire the greatest success in prostatic surgery must be enabled to select, from his experience and knowledge of that operation, which is best suited to each individual case, and must be able to perform with equal skill and dexterity any of the various manipulations which may be necessary. It is no longer believed that any routine method is applicable to all forms of prostatic obstruction. Every case is a law unto itself.

A complete prostatectomy is justifiable if performed early, before the individual is broken down in health and secondary complications have supervened. In early operations the mortality varies between 5 per cent. and 7 per cent. If this operation is performed in feeble, elderly patients, with long-standing obstruction and secondary complications, the prognosis is grave and the mortality ranges between 15 per cent. and 18 per cent. If the bladder is hopelessly disabled the result obtained by the operation is negative. Cases of this description are only suitable for suprapubic drainage. A partial suprapubic prostatectomy is indicated in such cases as where a valve-like lobe exists. A perineal operation is best suited to those cases where the lateral lobe has a tendency to progress toward the rectum or obstruct the urethra; when the growth has assumed large proportions and projects well backward toward the bladder, a suprapubic operation is the one of choice. Our views regarding prostatectomy are based on an experience with thirty-eight operations. Arteriofibrosclerosis is a counterindication for a radical operation.

Dr. Horwitz was surprised to hear Dr. Deaver allude to the well-known fact that the prostate gland consists of but two lobes, instead of three, as a new discovery. Reference to the literature on the subject will show that this view has been held by the majority of genito-urinary surgeons for years.

Attention was called to the fact that Freyer had no claim as the originator of the method of total suprapubic prostatectomy which bears his name. On consulting the *British Medical Journal* of August and September, 1901, it will be found that his position is assailed by both American and English

surgeons. Letters published by Nichol, Bruce, Clark, Mayo Robson, Eugene Fuller, and others all show that the claim to originality is unfounded.

Dr. Deaver: I would like to make one or two remarks relative to the suprapubic operation. I have done thirteen: eleven in elected cases, and two in cases in which I felt the patients could not survive. Both of the latter patients were subjects of catheterization life for a long time. I have done these operations with two deaths. If I had refused to operate in the two fatal cases, as I did for several days, I would have had eleven straight cases without a death; but, as I was not operating for statistics, but for the relief of humanity, I operated on these two, with the mortality mentioned.

With reference to the pouch at the neck of the bladder Dr. Horwitz has referred to, the literature shows that when the finger is introduced after the removal of the prostate, there is no sac. This is a point made by all who have done suprapubic cystotomy. Another point is the early date at which urination is re-established. In the last patient this took place in ten days. The shortest period in my experience has been six days.

Dr. Neilson agrees with my paper in detail; therefore, there are no questions to discuss.

Dr. Horwitz has given the mortality; I had the statistics, but unfortunately did not bring them with me. The mortality of the Bottini operation is 6 or 7 per cent.; of the perineal route 8, and of the suprapubic, 11 per cent. The suprapubic operation is being done by men who a little while ago refused to operate by this route, and the probabilities are that in a short time the mortality will compare favorably with that of the perineal and Bottini operations. I have all due respect for the differences of opinion in the use of the Bottini instrument and the lithotrite. The crushing operation for stone is one of the most elegant in surgery. It shows the refinement of execution of which one may well be proud. The difference between the use of the lithotrite and the Bottini is very considerable.

The middle lobe of the prostate is a myth. The text-books speak of this —and I regret that my text-book speaks of the middle lobe of the prostate. Every book written now is a compilation, with a scattering through it of one's personal experience. Such a thing as a middle lobe of the prostate does not exist.

I think Dr. Horwitz cannot tell whether he ploughs through the left lateral lobe or an adenomatous outgrowth, but probably the therapeutic results are just as good.

A CASE OF SUTURE OF THE HEART, WITH RECOVERY.[1]

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I AM indebted to Professor Keen for the privilege of operating upon and reporting the following case:

T. E., aged twenty years, colored, cook, was admitted to the Jefferson Hospital, February 29, 1904. He had been stabbed in the chest with a long, rusty pen-knife. The stab caused severe pain, but neither felled him to the ground nor caused him to feel faint. He walked without assistance to the hospital, a distance of one and a half squares, then began to feel weak, and was found crawling up the steps leading to the entrance of the hospital. After being disrobed a small wound was found just above the third rib, about one inch to the left of the left margin of the sternum. The wound was surrounded by an emphysematous swelling, and bled continuously, the stream of blood being accelerated by each expiration. The temperature was 97° F., and the pulse 80, empty and markedly irregular in volume and rhythm. He lay on the right side, breathing forty times per minute in a short, jerky manner. At times he would complain of a little pain and severe dyspnœa. He was sweating and very pale, and had vomited once. There was neither cough nor blood-spitting. Entire left chest was tympanitic on percussion, except for a small area around the wound. On auscultation the heart beats could be heard indistinctly; there was no bruit nor splashing sound. Under ether anæsthesia operation was begun about forty-five minutes after the infliction of the injury. Time of operation about forty-five minutes. Amount of ether, four ounces. An incision was carried along the second rib for four inches to the sternum, then down the left margin of the sternum to the fourth rib and outward along the fourth rib for four inches. The musculocutaneous flap was dissected back and the opening in the chest found between the third and fourth ribs, the knife having descended after passing through the skin. The exposure was then made more complete by continuing the sternal incision downward for two inches, and the triangular flap thus formed retracted. The third and fourth ribs were severed and forcibly turned toward the right, fracturing the costal cartilages near the sternum. The knife had passed through the pleural

[1] Read May 4, 1904.

cavity and wounded the anterior edge of the left lung. The lung had collapsed into the vertebral gutter, and the pleural cavity contained a large quantity of clotted and fluid blood. The opening into the pericardium was easily found by the spurting of blood with each pulsation of the heart; it measured about three-eighths of an inch. The pericardial wound was enlarged in the axis of the heart, and a penetrating wound of the anterior wall of the left ventricle found. This wound was parallel to the axis of the heart, nearer the auricle than the apex, and measured about three-quarters of an inch (larger than either the skin, intercostal, pleural, or pericardial wounds). Bleeding from this wound was free and continuous, whether more marked during diastole I am unable to state, as the heart was now beating very rapidly and resembled a quivering mass of muscle. The wound was closed with a continuous silk suture (six stitches), the sutures being inserted and tied without reference to diastole for the reason already stated. During the suturing the coronary artery was punctured by the needle, causing a profuse hemorrhage which was controlled by an additional suture. The pericardial and pleural cavities were cleared of blood, and the pleural cavity was irrigated with salt solution. The pericardial wound was sutured with a continuous silk suture, a small opening being left at the lower end for the passage of a gauze drain. No attempt was made to suture the lung (which was not bleeding) nor the parietal pleura. A gauze drain was also placed in the pleural cavity, gaining exit at the lower part of the primary incision. The severed ribs were approximated and the soft parts sutured with silkworm-gut. During the operation twenty-four ounces of salt solution containing adrenalin were injected into the circulation, and strychnine and atropine were administered subcutaneously. After the operation the foot of the bed was raised, external heat applied, the extremities bandaged and owing to the violent struggling of the patient morphine was given. Temperature, 100.4°; pulse, 150; respiration, 32.

Second day: Temperature, 103.4°; pulse, 130; respiration, 56. Dressing saturated with blood-stained fluid. Unproductive cough. Strychnine, whiskey, codeine. Delirious, subsultus, carphologia. Great pain in left chest, garrulous wound.

Third day: Temperature, 100°; pulse, 130; respiration, 46. Drains removed and replaced between the edges of the skin wound because of oozing of blood.

Fourth day: Temperature, 102°; pulse, 140; respiration, 52. Passage of air through the external wound has ceased. Ammonium carbonate and digitalis.

Fifth day: Temperature, 102°; pulse, 130; respiration, 60. Examination of the chest reveals dulness, rough breathing, and crepitant rales over the lower part. Blood erythrocytes, 3,630,000; leukocytes, 38,800; hæmoglobin, 58 per cent. The chest was explored with a needle, with a negative result.

Sixth day: Temperature, 102.8°; pulse, 140; respiration, 48. Pus under skin-flap, removal of several stitches.

Seventh day: Temperature, 103.6°; pulse, 140; respiration, 52. Cough continues; little pain.

Eighth day: Temperature, 103°; pulse, 150; respiration, 50. More profuse discharge of pus from the wound.

Ninth day: Temperature, 103°; pulse, 136; respiration, 48. Chest again explored for pus, with negative result.

Tenth day: Temperature, 103°; pulse, 128; respiration, 52. Prolonged paroxysm of coughing.

Eleventh day: Temperature, 103.4°; pulse, 130; respiration, 50. Remaining stitches removed.

Twelfth day: Temperature, 103°; pulse, 128; respiration, 50.

Thirteenth day: Temperature, 100.8°; pulse, 120; respiration, 40. Small slough separating from the skin-flap.

Fourteenth day: Temperature, 100°; pulse, 120; respiration, 40.

Fifteenth day: Temperature, 101°; pulse, 120; respiration, 28. Cough almost gone; never any expectoration. Thereafter the temperature, pulse, and respiration gradually fell until on the twenty-second day the temperature registered 99°, the pulse 98, and the respiration 20. Subsequently the temperature, pulse, and respiration remained below these points. On the thirty-fifth day the patient was allowed out of bed. On the fifty-sixth day the patient was discharged to return to the Out-patient Department for dressing, a small sinus leading down to the point where the ribs were severed still persisting; this sinus closed a few days later. The ribs have united firmly with little callous formation. The pulse varies between 80 and 90. Adventitious sounds have never been heard in the heart. The left chest expands very little on inspiration, and slightly roughened breathing, but no rales, can still be heard.

In olden times a wound of the heart was considered synonymous with inevitable and immediate death. Ellsberg[1] tells us that prior to the sixteenth century Hippocrates, Celsus, Paul of Ægina, Roland, and Lanfranc all mention heart wounds, and agree that they are invariably fatal. In the early part of the sixteenth century Fallopius[2] studied the subject with a view to recognizing the cavity injured, and Jacob Hollerius made the assertion that a wound of the heart was not necessarily fatal. Later in the century bullets, arrowheads, and other foreign bodies were found in the hearts of animals which had continued to run for some time after receiving their death wounds, and tales were told of men who during the intoxication of battle continued to fight after being stabbed through the heart.

Toward the end of the century Ambroise Paré[3] related the oft-quoted case of the Turin gentleman who received a rapier thrust below the left nipple during a duel, and who made several passes and pursued his slayer 200 paces before falling to the ground a corpse.

In 1642 J. Wolf[2] gave the first reliable account of the healing of a wound of the heart. Morgagni[2] (1761) stated that the cause of death in many cases was compression of the heart by the effused blood, a theory which has since been converted into a fact mainly by the observations of Rose and the experiments of Cohnheim. It is interesting in this connection to recall the fact that Morgagni died of rupture of the heart.

Sherman[4] says Desault laid down the rules for pericardotomy for empyema in 1798, and it took surgery ninety-eight years to pass from the pericardium to the epicardium across a space that is such only potentially.

Benjamin Bell[2] (1783) did not believe healing could occur. A. Richter[2] (1786) considered the probe dangerous, and insisted upon absolute rest and free bloodletting. Larrey and Dupuytren laid down many rules for treatment. During the seventeenth and eighteenth centuries papers would appear describing cases in which protracted periods intervened between the injury and dissolution.

In the beginning of the nineteenth century Jamain, Zannetti and others wrote on the viability of patients after heart wounds; but it was not until 1867, when George Fischer[5] published his classical study of 452 human cardiac wounds, that a firm foundation was laid for the evolution of the surgery of the heart. Fischer clearly established the fact that an individual might exist for hours or even days with a wounded heart; and that, indeed, in from 7 to 10 per cent. of the cases the wound heals and the patient actually recovers. Loison and Ollivier collected additional cases and corroborated the findings of Fischer. Ninni[6] states that according to the statistics of Jamain, Fischer, Lantenelet and Zannetti, death is instantaneous in 18 per cent. of the cases.

Up until the closing years of the nineteenth century the treatment of wounds of the heart consisted in the application of an occlusive dressing, absolute rest, ice or blisters to the precordium,

sedatives, such as veratrum viride, digitalis, opium, and venesection. Rose[7] advised paracentesis of the pericardium or pericardotomy to relieve compression of the heart the result of a wound. In 1881 John B. Roberts suggested the propriety of attempting to suture a wound of the heart. Later Konig and Delorme voiced the same opinion. The idea, however, was generally regarded as the wildest fancy of ultra-enthusiasts. Billroth declared that no surgeon who wished to preserve the respect of his colleagues would ever attempt to suture a wound of the heart. Riedinger (1884) considered the proposition below the dignity of mention. Tillmanns was convinced of the hopelessness of surgery when confronted with a bleeding heart. That the heart could be exposed and manipulated has been demonstrated by many experimenters. In 1894 Kronecker and Schmey[1] discovered that this organ might be punctured with but little interference with its action, except when a spot in the interventricular septum was touched, when immediate death ensued (Kronecker's co-ordination centre). In 1895 Rosenthal[1] essayed to treat an experimental wound of the heart by direct means—he used a tampon of iodoform gauze, and the dog survived. During the same year Del Vecchio[1] was the first to suture the canine heart with success. In 1896 Salomoni[1] and in 1897 Bode[1] successfully closed wounds of animal hearts by suture.

Since then there have been many experimental investigations into this subject. The conclusions of Elsberg,[1] which may be taken as representative of the findings of the experimenters, are as follows: That the heart could be grasped with the hands or forceps and gently compressed, with no appreciable difference in its action, and that it could be penetrated by a needle and knife, producing only a temporary irregularity of its action; that penetrating wounds produced during systole bled more than those produced during diastole, and that wounds of the ventricles produced during systole were larger than those produced during diastole; that oblique wounds bled less than perpendicular wounds; that wounds of the right ventricle were more dangerous than those of the left because the wall of the right ventricle was thinner, and also because the blood in the right side of the heart coagulated more slowly; that wounds of the heart heal kindly and that the cicatrix is usually complete in about

fourteen days; that interrupted sutures are better than continuous ones; that the tissue included in the suture always atrophied, and was replaced by scar; a smaller number of muscular fibres was included in the interrupted than in the continuous suture, thus producing less atrophy and less scar tissue; that superficial sutures were less likely to tear out than deep ones, and that the sutures should be inserted and tied during diastole because of the danger of their tearing out if tied during systole.

After the road had been paved by the knowledge that patients would often live long enough for operative interference, and that the heart would tolerate manipulation, two unsuccessful attempts were made in 1896 to close human cardiac wounds by suture, one by Farina, and one by Cappelen, and in 1897 Rehn published the first successful cardiorrhaphy in man. Including these cases and the one reported in this paper, I have been able to find 60 cases with the surprisingly high recovery rate of 38⅓ per cent.

Wounds of the heart may be produced by penetration from without, as gunshot or stab wounds, or by fractured ribs, and by foreign bodies from the œsophagus, stomach or bronchus. The heart may burst as the result of blunt force to the thorax or epigastrium with or without laceration of the pericardium, and it may rupture spontaneously (disease of myocardium or coronary artery, neoplasms, gummata, echinococci, abscess, aneurysm, etc.). That an attempt should be made to save as many of these cases as survive for a sufficiently long period of time after the accident needs no argument. Of the cases thus far recorded, 55 were stab wounds and 5 gunshot wounds. Two of the gunshot wounds recovered (Launay, Noll) and in two of the fatal cases the posterior wound was not sutured (Bufnoir, Stern). As late as 1903, however, Wolff[8] advises expectant treatment in gunshot wounds of the heart.

The symptoms are those of acute anæmia or of compression of the heart, depending upon the escape of blood into the pleural cavity or externally, or upon its retention in the pericardial sac. In some cases the patient may walk or even run for a considerable distance before falling to the ground. In a recent stab case in Philadelphia the patient was examined by a careful and competent surgeon, who regarded the wound in the chest as trivial because of the absence of

symptoms. The patient died within twenty-four hours and an autopsy revealed a punctured heart. On the other hand, a patient may present marked symptoms without a serious injury. During the past year I was called to the Pennsylvania Hospital to see a boy who had been stabbed in the cardiac region. He presented all the classical symptoms of severe shock. Under a local anæsthetic the wound was enlarged and found to be non-penetrating. His symptoms disappeared in a few hours. In any case there may be nervous symptoms, such as vertigo, syncope, convulsions, vomiting, or hemiplegia (Pirogoff[6]). The nervous phenomena are said to be more common in injuries to the left ventricle, and to be due to anæmia of the spinal cord. Respiratory difficulties are more marked in wounds of the right ventricle, but may be due to nervous influences, pneumothorax, or compression of the heart. Hæmoptysis is rare, even if the lung is injured.

When the blood escapes into the pleural cavity (the pleura was injured in 57 of the 60 cases, 95 per cent.) there will be in addition to the symptoms of anæmia the signs of a pneumohæmothorax. Palpation may detect the apex beat. A splashing sound indicates blood and air in the pericardium. A whizzing sound due to the presence of air in the pericardium is also described. In some cases a friction sound may be heard, and in others a bruit not unlike that heard over an aneurysm. The heart is apt to be irregular and in many cases the pulse is below 100°.

When the bleeding is external the stream may be continuous or in jets. Slight increase in cardiac dulness is to be expected. The respiration may be quiet until near the end, and physical examination of the lung may reveal little abnormal.

When the blood is confined to the pericardium the pulse is exceedingly feeble, and the apex beat can neither be felt nor heard. There may be a splashing sound disappearing with the filling of the pericardium, at which time the area of precordial dulness will be vastly increased. The patient is unconscious, and may regain his senses on providing an exit for the blood. Cohnheim[9] demonstrated by animal experiments that the pressure manifested itself first on the auricles, and the origin of the great veins, producing venous stasis; this may increase to complete obstruction to the flow of blood,

and is manifested by dyspnœa and cyanosis. The ventricles pump themselves dry, and the heart finally stops.

Death ensues as the result of anæmia, compression of the heart, or later from sepsis or functional incompetence. Izzo[10] reports a case of stab wound of the left ventricle which was discharged cured on the twenty-eighth day. A few hours later the patient died while lifting a heavy body, the scar having ruptured. The possibility of embolism from air or clot should also be thought of.

As may be seen from the foregoing, a diagnosis of injury of the heart may be difficult or impossible without exploration. The location of the external wound is no sure guide. Of the external wounds in the 60 cases thirty-nine were on the left side, between the second and seventh ribs, sternum and anterior axillary line, two were in the left midaxillary line, two to the right of the sternum, one in the epigastrium, one under the left costal arch, and fifteen not mentioned. It may be stated, however, that any penetrating wound in the precordium is almost sure to injure the pericardium or the heart. The amount of external bleeding may be profuse and may be spurting from a wounded intercostal or internal mammary, and may be absent in a punctured wound of the heart. Although justified by Rehn, the use of the probe is generally condemned. The statement that the blood coming from the external wound will be bright red when the left heart is injured and dark in color when coming from the right heart is not to be relied upon. Neither can the size of the wound in the heart be predicted with certainty by the size of the external wound nor the cavity injured by its location. In the present case, for instance, the cardiac wound was larger than the external wound, the knife probably entering the heart at the beginning of systole, and as the heart swung forward added an incised wound to the stab, and the location of the external wound suggested a wound of the auricle rather than the ventricle. Of the 60 cases, the left ventricle was wounded thirty times, with fifteen recoveries; the right ventricle twenty-one times, with seven recoveries; the right auricle once, resulting in death; the left auricle once, resulting in death; the apex three times, with one recovery, and in 4 the chamber wounded is not known. The size of the wound in the heart muscle varied from 0.5 cm. to 7 cm. The only safe procedure in doubtful

cases is to enlarge the wound, ascertain if it penetrates the chest wall, and if there be symptoms of hemorrhage or "heart tamponage," operate.

Although the cases operated upon after the lapse of several hours show a larger proportion of recoveries than those operated upon earlier, operation should be undertaken as quickly as possible after the injury. Of the 60 cases the time elapsing between the injury and the operation is not given in 29, 11 of which recovered. Of the remaining cases 16 were operated upon within one hour, with four recoveries, 1 in three and three-quarter hours with recovery, 4 between three and four hours with one recovery, 1 in five hours with recovery, 1 in six hours with recovery, 2 in eight hours with one recovery, 2 in twelve hours with one recovery, and 3 after twenty-four hours with two recoveries. This gives a mortality of over 76 per cent. for those operated upon within four hours, and a mortality of 30 per cent. for those after four hours. In looking over the causes of death in these cases, and leaving out of consideration 2 cases in which the interventricular septum was cut, it may be noted that of the cases operated on under four hours $35\frac{5}{7}$ per cent. died within twenty-four hours, and of those operated upon after four hours $33\frac{1}{3}$ per cent. died within twenty-four hours. An individual who survives a heart wound for a number of hours has a fair chance to recover with operation, and perhaps without operation, but of the cases which would succumb within a few hours many will be saved by prompt intervention.

The advice given by Rydygier,[11] to allow a knife which may be found sticking in the chest to remain until ready to suture, is questionable. It is easy to understand how the blade might produce further laceration of a beating heart.

An anæsthetic should always be employed unless the patient be unconscious. Ether was employed in the present case, because it was believed to be safer than chloroform. Of 4 cases in which ether was employed, 2 died, 1 on the table; of 11 cases in which chloroform was used, 4 died, 1 on the table, and of 11 cases in which no anæsthetic was given, 6 died, 2 on the table. Of 2 cases in which it is simply stated that an anæsthetic was administered, both died, 1 on the table.

The heart may be exposed by the extrapleural method or by the transpleural method.

Among those who have employed or who advise the extrapleural method are Rydygier, Pagenstecher, Napalkow, Wehr, Giordano, Podrez, and Nietert. Rydygier[11] advises a horizontal incision at the upper border of the third rib extending from the right of the sternum to the left costochondral joint, then obliquely downward along the outer ends of the costal cartilages as far as the fifth interspace. The third, fourth, and fifth costochondral articulations are divided, the sternum severed subperiosteally and the flap turned to the right. Giordano[6] suggests a vertical incision along the right margin of the sternum, with the division of the second and third cartilages; from each extremity of the vertical incision a transverse cut is made across the sternum, which is then divided and the flap turned to the left by luxating the cartilages. Podrez[6] begins an incision in the second left interspace one and one-half inches from the sternum, carries it as far as the midsternum, then downward to the level of the seventh cartilage and outward along the seventh rib. The sternum is divided at either end of the longitudinal incision and the flap turned to the left. Nietert[12] cuts from the right border of the sternum to one inch to the left of the sternum on a level with the third rib; a similar incision is made on a level with the base of the ensiform, and the left extremities of these incisions are united by a perpendicular incision. The fourth, fifth, and sixth ribs and the sternum are divided and reflected to the right.

Among the transpleural methods are those of Del Vecchio, Parlavecchio, Farina, Rehn, Cappelen, Parozzani, Giordano, Ramoni, Ninni, and Rotter. Del Vecchio[6] outlines an H-shaped incision; the vertical limbs correspond to the left sternal and mammary lines, and the horizontal portion to the fourth interspace. The fourth, fifth, and sixth ribs are resected, one flap being turned upward and the other downward. Farina, Rehn, Parlavecchio, and Cappelen resect one or more ribs. Parozzani[6] made a triangular flap, one side of which extended along the left margin of the sternum from the fifth interspace to the ninth rib, the second side from the upper end of this incision outward for 3 cm.; the sixth, seventh, and eighth ribs were cut and the flap turned outward. Giordano[6] incised in the

third interspace, sectioned the third and fourth ribs, turning the one upward and the other downward. Ninni advocates an incision along the sternum from the third to the sixth rib and the same length in the mammary line, joining these by a horizontal incision in the fifth interspace. The intervening ribs are cut and the flap turned upward. Rotter[12] cuts outward from the left border of the sternum along the third rib for two inches, then downward parallel with the left border of the sternum to the sixth rib, thence along the sixth rib to the sternum. The third, fourth, and fifth ribs are severed and the flap turned toward the right. He also advises the same procedure on the right side in cases in which the wound is on the right.[13] The opening was made in the right chest in 3 cases only (Watten, Ninni, Barth).

The extrapleural method will be indicated in cases in which the wound does not involve the pleura and in which the patient's condition is such as to sustain an operation protracted by the small opening in the chest wall and by the careful manipulations necessary to avoid the pleura. This will be rare; in 3 cases only did the pleura escape injury (Pagenstecher, Nietert, Gibbon). Rehn[6] thinks it advisable to incise the pleura to avoid negative pressure from aspirating air into the mediastinum, causing the blood to foam.

An atypical osteoplastic flap with the base toward the sternum either in the right or left chest, according to indications, and including as many ribs as may be necessary for proper exposure, usually from two to four, will probably be used in most of the future cases.

The wound in the lung ceases to bleed as a rule when the lung collapses and needs no treatment; if bleeding it may be sutured.

The wound in the pericardium is enlarged in the axis of the heart by tearing or cutting, and the sac cleared of blood with the finger. In several of the cases attention is called to the fact that the blood coagulated behind the heart. In experimental work Ricketts[14] found that collapse of the lung would congest the heart, and that exposure caused it to dilate because of the lack of support of the pericardium and chest wall.

The bleeding from the heart may be controlled by a finger, and the heart may be steadied by the fingers, by forceps, or by sling

sutures. In the present case a suture was inserted at the upper end of the wound, tied, and used as a tractor for the insertion of the remaining stitches. In 2 cases the sutures tore out (Walker, Zulehner). Should the heart cease to beat during the manipulation it should be sutured quickly and massage performed. The use of gauze packing will probably never be indicated; to be efficient it must be firm, and to be firm it must exert injurious compression. A round curved intestinal needle is generally recommended. Silk was employed in 26 cases with two recoveries, catgut in 9 with five recoveries, celluloid in 1 with recovery; and in 24 the suture material is not known. The highest number of sutures employed was eleven. Silk has the advantage of being easier to manipulate and more certain to hold. It has been proven that catgut will last long enough for the repair of the wound. Silk was used in the present case because of the fear of untying of the knots by the constant gyrations of the heart. The continuous suture is mentioned in 3 of the cases, with two recoveries. It may be applied more rapidly than the interrupted, and presents fewer knots on the surface of the heart. The sutures are inserted at intervals of one-eighth of an inch deeply into the muscle, but must not penetrate the endocardium. In dogs Sherman[4] found it a difficult matter to penetrate the endocardium with an ordinary curved needle. After tying, the ends should be cut off one-quarter of an inch from the knot to guard against untwisting. Diastolic suture is difficult and unnecessary. One would have to rehearse with a rapidly ticking metronome for a long time before he would be able to accomplish the feat. In dogs Ricketts[14] and Sherman[4] found it impossible. Although Larrey showed many years ago the possibility of recovery after a severed coronary artery, it has been asserted repeatedly that this accident is always fatal. Ricketts demonstrated on animals that either coronary artery may be ligated at any point without death resulting. Of the 4 cases in which the coronary was injured, only 1, the present case, recovered.

Any blood which may be in the pericardium is now removed by the finger or sponge, and the pleural cavity cleared of fluid and clots. The pleura and pericardium may be sutured or both cavities may be drained. Wolf[8] shows that there is no retention of secre-

tion in the pericardium when it is closed. He explains the presence of fluid behind a tampon in Watten's case by the massage of the heart by the gauze. He says the cardiac wound heals quickly and is not infected, the instrument having been cleaned on its way through the chest wall, and he strongly advises suture of the pericardium without drainage. The dangers of the drain are the same here as elsewhere, *i. e.*, infection and adhesions. Of the 22 cases in which the pericardium was drained, 2 died within a few hours, and of the remaining 20, 10 (50 per cent.) recovered; of the 15 in which the pericardium was closed without drainage, 1 died in fifteen minutes; and of the remaining 14, 7 (50 per cent.) recovered. The question may still be regarded as an open one. In a future case I think I should close the pericardium and drain the pleura through a stab in the back, as the latter cavity is difficult to clear of blood quickly.

Of the 23 cases that recovered after operation 11 are known to have been complicated with some form of infection; in only 1 case is uncomplicated recovery stated. Of the 37 deaths, 16 occurred within twenty-four hours from shock and hemorrhage, and in 2 of these the interventricular septum had been punctured; 3 died on the second day, 2 from the infection of the pleura and 1 unknown; 4 died on the third day, 2 from infection and 2 unknown; 2 died on the fourth day, both from infection, and in one of these the tricuspid valve had been injured; 3 died on the fifth day from infection, and 1 on the sixth, 1 on the fourteenth, 1 on the nineteenth, and 1 on the twenty-second day, all from infection; in 5 cases the time and cause of death are unknown.

I append a table of the reported cases.

Table of Cases of Cardiorrhaphy.

Operator and year.	Location of external wound.	Chamber wounded and size of wound.	Time between injury and operat'n.	Anæsthetic.	Suture.	Drainage	Result and remarks.
1. Farina, 1896	Just above margin of left 6th rib, near sternum.	R. v., ¼ in.	Immediately.	.....	3 silk sutures.	Suture of pericardium.	Death 6th day; bronchopneumonia.
2. Cappelen, 1896	4th left intercostal space in midaxillary line.	L. v., $^{4}/_{5}$ in.; left coronary artery sever'd	1 hour.	.....	.....	.....	Death 2½ days; pericarditis.
3. Rehn, 1897	4th left intercostal space near sternum.	R. v., 1½ cm.	24 hours.	Ether.	3 silk stitches	Drain of pleura and pericard'm.	Recovery; empyema.
4. Parozzani 1897	7th left intercostal space in midaxillary line.	L. v., ¾ in.	5 hours.	None.	2 silk sutures.	None.	Recovery.
5. Parozzani 1897	3d left intercostal space.	L. v., $^{3}/_{5}$ in.	½ hour.	None.	2 silk sutures.	....	Death 24 hrs.; interventricular septum cut, also pleurisy on l. side.
6. Fummi, 1898	Under left nipple.	Apex, cavity not opened.	Several hours.	.....	1 silk suture.	.....	Recovery; empyema.
7. Ninni, 1898	5th left intercostal space, mammary line.	L. v., 1 in.	Immediately.	None.	2 silk sutures.	No drain pericard'm.	Death on table.
8. Parlavecchio, 1898	5th left intercostal space.	L. v., 1½ in.	8 hours.	Chloroform.	.....	No drain.	Recovery.
9. Giordano, 1898	2d left intercostal space.	L. a., $^{4}/_{5}$ in.	½ hour.	None.	4 silk sutures.	Drain of pleura.	Death 19th day; empyema and abscess r. lung.
10. Williams, 1898	.....	L. v., not penetrating.	.....	.....	.....	.....	Recovery.
11. Nicolai, 1899	4th left intercostal space, mid. between sternum & nipple.	R. v.	1½ hrs.	Yes.	.....	.....	Death after 12 hours.
12. Tuzzi, 1899	4th left intercostal space.	2 wounds, non-penetrating.	.....	None.	.....	Drain of pleura and pericard'm.	Death 22 days; empyema and pericarditis.
13. Longo, 1899	5th left intercostal space $^{2}/_{5}$ in. internal to nipple.	L. v.	Immediately.	None.	3 silk sutures.	No drain of pericard'm.	Death in 15 min.
14. Ramoni, 1899	3d left costal cartilage $^{4}/_{5}$ inch from sternum	R. v., 2 w'ds, 1 non-penetrating.	.....	None.	4 silk sutures.	No drain of pericard'm, pleura dr.	Recovery.
15. Marion, 1899	Shot through breast.	R. v.	.....	.....	Catgut sutures.	.....	Death.
16. Rosa, 1899	5th intercostal space.	L. v., $^{3}/_{5}$ in. not certain it penetrat'd	.....	None.	1 silk suture.	Drain of pleura and pericard'm.	Recovery.
17. Horodynski, 1899	.....	R. v., 1½ cm.	.....	.....	.....	.....	Death.
18. Maliszewski, 1899	....	.....	.....	.....	.....	Drain of pericard'm.	Death; suppurative pericarditis.
19. Maliszewski, 1899	.....	.....	.....	.....	.....	Drain of pericard'm.	Death; suppurative pericarditis.
20. Bufnoir, 1899	6th left intercostal space.	R. v., gunshot, 22 calibre.	.....	.....	.....	.....	Death; perfor'n of ventricle, and ant. opening only had been sutured
21. Kosinki, 1899	.....	R. v.	.....	.....	.....	.....	Recovery.
22. Pagenstecher, 1899	4th left intercostal space beneath nipple.	L. v., near apex, $1^{2}/_{5}$ in.	2 days.	None.	4 celluloid sutures.	Drain by threads from myocardium.	Recovery.
23. Nanu, 1900	3d intercostal space 4 cm. from edge of sternum.	R. v., 2 cm.	.....	.....	2 interrupted sutures. of silk.	.....	Death 5th day from infection of pericardium and pleura.
24. Masselli, 1900	Below and internal to left nipple, cutting 6th rib.	L. v., near apex.	1½ hrs.	None.	2 sutures	Drain of pleura.	Death in 12 hrs.
25. Foutan, 1900	6 wounds between 3d and 7th ribs in cardiac region.	L. v., 12 mm.	6 hours.	Chloroform.	2 continuous catgut sutures.	No drain, suture of pericard'm.	Recovery.
26. Carnoble, 1900	.....	Apex.	.....	.....	Catgut.	.....	Death in 5 days; empyema.
27. Nietert, 1901	Right of sternum.	R. v.	2 hours.	None, unconscious.	2 silk sutures.	Drain of pericard'm.	Death in 33 hrs.
28. Vaughan, 1901	5th left costal cartilage divided.	L. v., 2½ cm.	¾ hour.	Ether.	7 continuous silk sutures.	.....	Death on table.
29. Ninni, 1901	Left of sternum.	R. a.	.....	.....	.....	.....	Death in 4 days; sepsis.
30. Mignon et Sieur, 1901	.....	R. v.	.....	.....	.....	.....	Death.

Operator and year.	Location of external wound.	Chamber wounded and size of wound.	Time between injury and operat'n.	Anæsthetic	Suture.	Drainage	Result and remarks.
31. Fontan, 1901	.....	L. v.	.....	.....	Catgut sutures.	Suture of pericard'm.	Recovery; empyema.
32. Brenner, 1901	Left of sternum near 6th cartilage.	R. v., 7 cm.	Following day.	Yes.	.....	.....	Death on table.
33. Watten, 1901	4th right intercostal space.	R. v., 3½ to 4 cm.	.....	.....	.....	Drain of pericard'm.	Recovery; right pleura wounded, pneumothorax.
34. Lastaria, 1901	.....	L. v.	.....	.....	.....	.....	Death in 12 hrs.
35. Zulehner, 1901	.....	L. v., ½ cm.	.....	.....	.....	.....	Death on table.
36. Zulehner, 1901	.....	R. v. 1 cm.	.....	.....	Catgut sutures.	.....	Recovery.
37. Stern, 1901	.....	L. v., pistol wound.	.....	.....	Unable to suture posterior wound.	Gauze tampon.	Death in 30 min.
38. Pagenstecher, 1901	In heart region.	L. v., left coronary artery injured.	.....	.....	.....	Drain of pericard'm.	Death in 5 days; pus in pleura and pericard'm.
39. Ninni, 1901	.....	L. v.	.....	.....	.....	.....	Recovery.
40. Walker, 1901	.....	L. v., 8 stab wounds.	.....	.....	11 silk sutures.	Drain of pericard'm.	Death in 24 hrs.; pyothorax and pyopericardium.
41. Walker, 1901	Under left costal margin.	Apex r. v.	.....	.....	Suture tore out repeatedly.	Gauze tampon.	Death in ½ hr.; hemorrhage.
42. Nitert, 1902	Left 6th intercostal space to right of papillary line.	L. v., 2 cm.	14½ hrs.	Chloroform.	2 silk sutures.	Drain.	Recovery; empyema.
43. Launay, 1902	Left nipple.	L. v., ant. & post. walls, pistol shot.	3½ hrs.	Chloroform.	Catgut suture in each wound.	Drain of both cavities.	Recovery; uncomplicated.
44. Gibbon, 1902	Left precordium.	R. v., 1.5 c.m.	½ hour.	Chloroform.	1 catgut suture.	.....	Death on table.
45. Hill, 1902	5th intercostal space a little to right of nipple.	L. v., ⅜ in.	8 hours.	Chloroform.	1 catgut suture.	.....	Recovery.
46. Stewart, 1902	4th intercostal space ½ inch left of sternum.	L. v., ⅝ in.	Immediately.	Ether.	6 catgut sutures.	.....	Death in 3 days.
47. Pomara,[15] 1902	.....	.....	.....	.....	.....	.....	Death in a few days.
48. Milesi,[16] 1903	Upper border 6th rib 1.5 cm. from left border of sternum.	R. v., 1.5 c.m.	½ hour.	.....	Silk suture.	Drain of pleura and pericard'm.	Death in 15 hrs.; punct. interventricular septum.
49. Giordano[17] 1903	2 wounds, 1 in 4th and 1 in 6th intercostal space.	L. v., 2 cm. long.	.....	.....	2 silk sutures.	Drain.	Recovery; necrosis of ribs.
50. Giordano[17] 1903	6th intercostal space 1 cm. left of nipple.	L. v., near apex.	.....	.....	2 silk sutures.	Drain.	Death in 1½ hrs.
51. Vinci,[18] 1903	5th left intercostal space in parasternal line.	L. v., 6 cm., coronary artery injur'd during suturing.	About 12 hours.	Chloroform.	3 silk sutures.	No drain of pericardium, drain of pleura.	Death 2d day; pneumonia and pleurisy.
52. Janari,[19] 1903	3d left intercostal space bet. ant. axillary & mam. lines.	L. v., 2 cm.	¾ hour.	Chloroform.	3 silk sutures.	Drain of pericard'm.	Death in 74 hrs.; pleurisy.
53. Barth, 1903	Epigastrium and through ensiform.	R. v., 2 cm.	Soon.	Chloroform.	4 silk sutures.	Drain of pleura and pericard'm.	Death 4th day; sepsis triespid valve injured.
54. Barth, 1903	4th left intercostal space 3 cm. from sternum.	L. v., ½ cm.	Soon.	Chloroform.	3 silk sutures.	Suture of pericard'm.	Recovery, with pleurisy.
55. Barth, 1903	3 cm. above and inside nipple.	R. v., ¾ cm.	.....	Chloroform.	5 silk sutures.	Suture of pericard'm.	Recovery, with pleurisy.
56. Schwerin[20] 1903	4th left intercostal space.	R. v.	¾ hour.	.....	3 silk sutures.	Drain.	Recovery; pyopericardium.
57. Noll,[21] 1903	Revolver wound 5th left intercostal space.	L. v., ant. wall sutur'd, post. not ment'n'd.	1½ hour.	.....	.....	Drain.	Recovery; empyema.
58. Wolff,[22] 1903	.....	R. v., 2 cm.	.....	.....	.....	Drain.	Death in 14 days; empyema.
59. Stude,[23] 1904	Left breast.	L. v., 1 cm.	1¾ hour.	.....	Silk suture.	.....	Death on table.
60. F. T. Stewart, 1904	2d intercostal space 1 inch left of sternum.	L. v., ¾ in., coronary artery injured during suturing.	¾ hour.	Ether.	6 continuous silk sutures.	Drain.	Recovery; pneumonia and infection of wound in chest wall.

Cases 1 to 46 inclusive and Cases 53, 54, and 55 are taken from Hill[10] and Wolff.[8]

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THE TREATMENT OF PENETRATING WOUNDS OF THE HEART.[1]

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It is difficult for anyone to speak with authority born of experience on the treatment of penetrating wounds of the heart, since opportunity for repair of such injuries is so rare. Dr. Stewart has presented a table of sixty cases, which are all that have been recorded in surgical literature, and but two surgeons have operated on as many as three cases. My own excuse for attempting to discuss this most important subject is the interest I take in it, due to an unsuccessful effort to suture a stab wound of the right ventricle. I have reported this case in the *Philadelphia Medical Journal*, November 1, 1902. This was the fourth case operated upon in this country.

A Fellow of this College, Dr. John B. Roberts, was one of the first to suggest the advisability of suture in cases of penetrating wounds of the heart. This he did in a paper read before the Anatomical and Surgical Society at Brooklyn, in 1881. It was, however, not until 1896 that the first case of operation for this condition was reported. Dr. Stewart's table, perhaps, does not present all the operations, since no doubt there are a number of unsuccessful cases which have never been put on record. The results obtained in these cases, twenty-three recoveries, certainly teach us that the former surgical attitude toward penetrating wounds of the heart was altogether wrong, and that we should always attempt their repair whenever the opportunity presents itself. It is evident that certain cases are more hopeful than others. Those patients who are able

[1] Read May 4, 1904.

to survive the injury for some hours or even a day are much more likely to recover after operation than those who immediately pass into unconsciousness and present other symptoms of cardiac compression or those of excessive loss of blood. The repair of gunshot wounds of the heart has not been so successful as that of stab wounds, and probably one reason for this is the fact that in the former there are often two wounds. Peyrot says that 17 per cent. of gunshot wounds are double, while only 1.2 per cent. of stab wounds are. In this connection it is interesting to refer to a case which Trendelenburg reports, in which a bullet was lodged in the right ventricle, and the patient recovered without operation. The bullet could be seen with the fluoroscope and its movements observed. Later, it became fixed in the heart wall. Strange as it may seem, there are nineteen such cases on record where bullets have lodged either in the heart cavities or wall, and the patients have lived for varying periods. Rickets, in his most exhaustive study of the surgery of the heart, refers to the frequency with which the heart has been found to be most unexpectedly the seat of injury due to its anomalous situation upon the right side or even within the abdomen.

As one would expect to find, the Italian surgeons are those who have contributed most largely to the literature on this subject. The Germans also have reported a large number of the cases. Williams, of Chicago, in 1898, reported the first successful case in America. Nietert, of St. Louis, in 1901, lost a patient thirty-three hours after a successful suturing of the heart, from suppression of the urine, but was able a year later to report a successful case, the second in this country. Hill, of Birmingham, also has recently reported a recovery after operation. The case reported by Stewart to-night is therefore but the fourth successful one in America.

Regarding the technique of repairing penetrating wounds of the heart, it is impossible to lay down any fixed rule, since it must vary largely with the general condition of the patient and the situation of the wound. In my own unsuccessful case I think I lost valuable time in scrubbing the field of operation after the patient was put upon the table, but I should have felt very remorseful had he recovered from his injury and died from sepsis. From a study of the reported cases, however, I would conclude that in patients who

are in a desperate condition, the part of wisdom would to expose and repair the heart with the least possible delay. In patients, on the contrary, who have survived the injury for a number of hours or who are not in an immediately dangerous condition, sufficient time should be taken to thoroughly disinfect the field of operation. The question of employing an anæsthetic is one about which there has been considerable discussion, and here again it is not possible to form any fixed rule, since in the completely unconscious cases an anæsthetic is unnecessary, and in the conscious cases it is as strongly indicated. That the manipulation and suture of the heart itself is not painful is shown in Nietert's first case, where the patient regained consciousness during the introduction of the sutures and conversed with the surgeon during the closure of the wound. Although an anæsthetic has its disadvantages at such a time. the struggling resulting from the exposure of the heart without an anæsthetic carries with it the danger of loosening a clot which may have temporarily occluded the opening in the cardiac wall. This danger can be illustrated in a case of penetrating wound of the heart which healed without operation, and in which the patient died some time later after violent exertion, producing a dislodgement of the hæmostatic clot. The incision and the nature of the flap must vary with the seat of the injury and also with the promptness with which the heart must be exposed. The tendency of most operators in their first cases has been to resect the necessary amount of bone and cartilage to thoroughly expose the wound. It is the judgment of the most experienced, however, that a deliberate osteoplastic flap with its base at the sternum or beyond the costocartilaginous articulations is to be preferred. Having found the wound in the pericardium and opened it, if bleeding is taking place a hurried digital examination of the heart should be made, and when the opening is found the tip of the finger should be inserted into it and carefully kept in position in order to prevent the escape of more blood. In some instances it has been possible to introduce the sutures while the finger was still inserted in the opening. If this cannot be done two procedures may be followed: that of introducing traction sutures for the purpose of lifting up the heart, and the other, of placing two fingers behind the heart so as to bring it into the wound. Suture during diastole

has been strongly recommended by some. Those, however, who have had experience in closing such wounds seem to agree that it makes little difference when the suture is introduced. It seems equally unnecessary to carefully avoid entering the heart cavity with the suture, since a number of experimenters have shown that it is no easy matter to introduce the suture to that depth. It makes little difference what suture material is used, although personally I should prefer chromicized gut introduced on a small, full curved intestinal needle. When catgut is used instead of silk, however, care must be taken to make at least three knots, so as to overcome the danger of loosening. Having closed the anterior heart wound a rapid search should be made for a wound of exit; if none is found the pericardium should then be cleared of clots by damp sponging, and the pericardial wound closed with or without drainage. If the pleura has not been involved in the primary injury it is apt to be opened in the exposure of the heart. The question of drainage is one about which there is also considerable dispute. If the operator has been able to thoroughly cleanse the field of operation and believes that there has been no contamination during the operation, I can see no more reason to drain either the pericardium or the pleura than there would be after opening the abdomen for an intra-abdominal hemorrhage. If, on the contrary, the operation has been done hurriedly and without thorough preparation, then I think that drainage of both the pericardial and pleural cavities should be established. If it should be desired to drain the pleura and not the pericardium, I believe that this had best be done at a point distant from the field of operation, since in this way drainage at the most dependent point can be obtained, and, in case suppuration takes place, infection of the pericardium possibly avoided.

MEDICAL AND SURGICAL CONSIDERATIONS IN PYOPERICARDITIS, WITH REPORT OF CASES.[1]

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MEDICAL ASPECTS, BY J. A. SCOTT, M.D.

PERICARDITIS is usually divided into primary and secondary varieties. Primary pericarditis is so rare that it can be dismissed without further consideration. Secondary pericarditis, or that concurrent with or sequent to other diseases, is that commonly met with in practice. This paper deals only with purulent pericarditis of this type.

FREQUENCY OF PERICARDITIS. This is a difficult matter to decide, for statistics vary considerably. Anders' figures, collected to show the frequency of cardiovascular lesions in croupous pneumonia, show in 275 autopsies the presence of acute pericarditis in 14 cases, or 5 per cent., and chronic pericarditis in 11 cases, or 4 per cent. Riesman, in a study of adhesive pericarditis, analyzed 778 necropsies in the Philadelphia Hospital, and found 60 cases of pericarditis, 7.7 per cent.; 20 of these were adhesive; very few were purulent. Breitung, collecting 324 cases of pericarditis at the Berlin Charité, found the following figures: serofibrinous cases, 108; hemorrhagic, 30; purulent, 20 (6.1 per cent.); secondary tuberculous, 24; primary tuberculous, 2; partially adhesive, 111; totally adhesive, 23; ossifying, 2. The proportion of cases of pericarditis occurring

[1] Read May 4, 1904.

in autopsy work is given in von Ziemssen's *Handbook* by various authorities as follows: Duchet, 15.1 per cent.; Chambers, 16.2 per cent.; Willigh, 14.1 per cent.; Taylor, 12.5 per cent. G. E. Still, in an article on "Suppurative Pericarditis in Children," finds in 769 autopsies 28 cases of pericarditis, apparently due to pyogenic infection. Twenty-three of the 28 cases occurred in children under three years of age, and 24 of the 28 showed empyema, thick lymph on the pleura, or acute pleurisy. In 13 cases in which the fluid was examined bacteriologically, 11 were due to pneumococcal infection; of the remaining 15, a large proportion were due to the pneumococcus—empyema, pneumonia, and meningitis being associated.

Analyzing the mortality statistics of the Pennsylvania Hospital, since the opening of the Ayer Clinical Laboratory, I have found 40 cases of pericarditis in 552 autopsies. Ten were obliterative in type; in 13 the exudate was serofibrinous; in 16 (40 per cent.), purulent; in 1 hemorrhagic. The great discrepancy will be observed at once between these statistics and those of Breitung, in which were found but 20 cases of purulent pericarditis in a total of 324 cases of all varieties. Clinically these 40 cases were etiologically as follows: tuberculous, secondary, 7; rheumatic, with acute or chronic endocarditis, 5; aneurysm, cardiac and abdominal, 2; nephritis, general streptococcus septicæmia, cirrhosis of liver, and secondary anæmia, 1 each; and lobar pneumonia, 22. In all these 22 cases pleurisy coexisted with the pericarditis; in 6 cases both the pericarditis and pleurisy were fibrinopurulent; in 4 each process was fibrinous; in 3 cases purulent pericarditis was associated with plastic pleurisy, and in 3 cases the pleurisy was fibrinopurulent, while the pericarditis was fibrinous. In 2 cases plastic pleurisy and adhesive pericarditis coexisted, and in 2 others the pericarditis was purulent, while the pleurisy was serofibrinous. In the remaining case the pericarditis was serofibrinous, and the pleural effusion was bloody.

Again, recognizing the great frequency of purulent pericarditis in croupous pneumonia, I have gone over the total number of autopsies upon that disease, and find them to total 76 cases of croupous pneumonia and 5 of bronchopneumonia. In 38 cases of croupous pneumonia, or exactly 50 per cent., pericarditis has been found: in 20 cases, or 52.6 per cent., the exudate was serofibrinous; in 17, or

44.7 per cent., the pericardium contained a purulent exudate; in one case the pericardium was obliterated (chronic adhesive pericarditis). In none of the bronchopneumonias (non-tubercular in origin) was pericarditis present.

Etiology. Purulent pericarditis is seen in pyæmic diseases—the streptococcal and staphylococcal infections—as a result of empyema, and in perforation of abscesses in the neighborhood of the pericardium; cancer of the œsophagus, cardiac and aortic aneurysms, etc. It is, I believe, commoner during lobar pneumonia than is recognized, and, as the autopsy records at the Pennsylvania Hospital will show, must be responsible for the death of the patient in not a few cases. The filling of the pericardium with exudate during croupous pneumonia is, I believe, not infrequently overlooked. The diagnosis, however, under such circumstances is a difficult one, as will be detailed later.

Bacteriology. The variety of micro-organisms found in purulent pericarditis depends upon the primary infection producing the disease. The purulent form is most commonly caused by streptococci, staphylococci, bacilli coli communis, pneumococci, and the tubercle bacilli. There is no reason why the gonococcus and the typhoid bacillus should not play a part in purulent pericarditis. When a connection is established between the pericardium and the œsophagus or the lung numerous saprophytic micro-organisms may be found.

Symptomatology. Symptoms are often difficult to interpret correctly, mainly because of the fact that pyopericarditis is commonly only one part of the symptomatology of such serious illnesses as a general pyæmic state, abscesses of the mediastinum, or pneumococcal and other infections.

General Appearance. The patient usually presents an anxious expression and dusky pallor of the skin, with dilatation of the alæ of the nose. Dyspnœa is usually present, at times extreme. The pulse in these cases is quick, with a sharp rise under the finger, rapid and of low tension; very unlike the pulse of an acute infection. At times the large veins of the neck are much distended.

Pain. Common usually over the region of the heart, but of moderate severity; again, it may be intense and agonizing, radiating

over the left chest and down the left arm. It is commonly increased by pressure in the epigastric region. During the development of a purulent pericarditis, and while friction rub is present, the pain is apt to be more severe, and disappears to some extent with the presence of a large effusion. In many cases pain in the epigastric region is an important symptom; this is accompanied with a sense of constant fulness in the same area; nausea and vomiting are apt to be present in this type. The vomiting is bile-tinged in some cases.

Dyspnœa is usually present and is disproportionate to the amount of pulmonary involvement and the rate of pulse.

Cough. Often dry, frequent, and distressing. Often non-productive. If the pericarditis is associated with pneumonia the sputum may, of course, be blood-tinged. Aphonia and dysphagia may be present.

Temperature. Fever is, of course, complained of to some extent. The temperature is at times but slightly elevated, though the pericardium may be filled with pus. The pulse is weak, small, and may present an intermittency known as the pulsus paradoxus, a condition in which the radial is either greatly weakened or altogether obliterated during inspiration. Delirium in pericarditis of all forms is common. At times it is active, at other times it is low. It is especially to be found in the rheumatic cases with hyperpyrexia.

Physical Signs. Purulent pericarditis at times develops without the ordinary physical signs of the disease being present, or at least discoverable; that is, though carefully sought for, the presence of friction rubs over any portion of the precordial area cannot be detected. In many cases pericardial friction rubs antedate the presence of effusion. This is certainly so in the serofibrinous cases. They are heard most commonly in the second, third, and fourth interspaces to the left of the sternum, perhaps more frequently over the base of the heart than at the apex. They are close to the ear, rough in character; sometimes both coarse and fine sounds can be heard. Heard with the systole and diastole of the heart and not always consistent in length or duration. Pressure increases their intensity. Their inconsistency is their characteristic. Change in position alters the character and time of the sound. At times these

rubs can be heard over a large area of the chest, under some circumstances as far as each nipple and down into the epigastric region. Friction sounds, of course, disappear with the presence of effusion into the pericardial sac. Under such circumstances a distinct alteration in physical signs occurs. The apex beat, which heretofore was in its normal position, becomes slightly elevated, perhaps to the fourth space, or may disappear altogether. It may, however, still be palpable in the fourth space. The sounds at the apex are distant and weak. With a normal endocardium no valvular murmurs are heard. Frequently, however, in these cases the causative factor in producing the pericarditis also has caused an endocarditis. Not infrequently the two conditions coexist. Valvular murmurs are therefore to be heard at the areas affected. Coincident with the disappearance of the apex beat occurs an increase in the precordial area of dulness. This is especially noticeable upward to the left of the sternum, perhaps to the second rib and in the fifth and fourth spaces to the right of the sternum. Impaired resonance or dulness discovered in the fifth or fourth interspaces to the right of the sternum, with decided increase of the left border of the cardiac outline and disappearance of the apex beat with weak or feeble heart sounds, is certainly extremely suspicious of fluid in the pericardium. Under such circumstances other physical signs are apt to be present. Owing to the presence of large quantities of fluid in the pericardial sac there is compression of the left lung, especially the lower lobe, in the axillary region. Here at times can be detected a hyperresonant note. The same pressure frequently will produce an impaired or dull note at the extreme base of the left lung. On auscultation at this point the breath sounds may be bronchovesicular or bronchial This is a pressure consolidation. With physical signs such as these no hesitancy should be felt in inserting a needle into the fourth or fifth space near the left sternum, especially if the absence of the apex beat is demonstrated. In not a few cases fluid will be secured by exploratory puncture in the fourth or fifth right intercostal space. In some cases, however, the physical signs are extremely difficult of interpretation, owing to the presence of complicating conditions. Thus not infrequently in croupous pneumonia there is fluid both

in the chest and in the pericardium. Thus in Case II. an empyema of very considerable size dislocated the heart to the left so that the impulse was visible in the fifth interspace one inch outside the mid-clavicular line. With the evacuation of the empyema the pericardial condition was entirely overlooked, and this probably resulted in the man's demise. In two other cases under observation (Case IV. and one not reported in this paper) there was visible pulsation in the second and third interspaces to the right of the sternum, with the development of (in Case IV.) an enormous shield-like area of dulness between the two nipples, with absolutely clear breath sounds at the bases and axillary region on both sides. These physical signs could only therefore be explained by the supposition that the right side of the heart was adherent to the pericardium or that its pericardial effusion had displaced the heart *in toto* to the right. In the unreported case spoken of, adhesions between the lung and the pleura on the left side were so dense that the presumption is that the suspected pericardial effusion was behind both the lung and the heart, which in turn was displaced considerably to the right.

Temperature. The range of fever in purulent pericarditis is less than one would anticipate. As previously stated, the presence of pus in the pericardium will at times distinctly modify an already pre-existing high temperature. In the first case studied, while the pericardium was filled with the purulent exudate, the temperature was subnormal for a week. In Cases II. and III. the temperature range with the pus in the pericardium was between 98° and 100° F., and 100° and 102° F., respectively. In Case IV. the temperature was high. It is difficult to state the exact time of the onset of the pericarditis.

Pulse. The pulse is weak and of low tension from the start, irregular at times; in some cases influenced by respiration, either partially or totally obliterated. The paradoxical pulse was present in Case I.

Blood. A leukocytosis is produced in all cases. If, however, the pus has been present for any length of time in the pericardium, the leukocytes have a tendency to fall again toward, but do not reach, the normal. In cases with pre-existing leukocytosis—and this is found in practically all cases of purulent pericarditis—a hyper-

leukocytosis is added. In Case I. the leukocytes jumped from 6600 on the tenth day to 21,000 on the sixteenth day, and fell slowly to 17,050 on the twenty-eighth day. Three blood cultures were negative. In Case II. a leukocytosis of 22,700 was present on the fourteenth day. The pericardium at that time was probably full. On the twentieth day, with both pericarditis and empyema present, the leukocytes were 12,000. The blood was not counted after operation; no blood cultures were taken. In Case III. the leukocytes on the sixth day were 16,400; on the tenth day, 30,800. At this time pericarditis was diagnosed. Blood cultures on the third and fifth day were negative, but a positive culture was found on the tenth day after pericarditis was present. In Case IV. no cultures or counts were made. The urine is much reduced in quantity in all cases; usually contains albumin, no sugar, and on microscopic examination shows rather numerous casts; in our case granular casts have predominated, though hyaline were also found. The urea output in Case II. (the fatal case) was 30 grm. in the twenty-four hours.

In conclusion, in pneumonia the cardiac outline should be carefully mapped out at the onset. Any changes either in the size or position of the heart should carefully be watched for. With the development of an empyema more rigid scrutiny of the heart should be insisted upon. The presence of dulness in the fifth right interspace or the dislocation of the heart either to the right or to the left side without a coexisting empyema, should lead one to suspect the presence of fluid in the pericardium, and careful selection of the spot for exploratory puncture should be made.

Diagnosis. Pyopericarditis is to be diagnosed by the following signs: The presence of a primary infection capable of producing pus; the development of a rapid, weak, and at times intermittent pulse, cough, and dyspnœa; pain over the heart and epigastric region of severe nature; a temperature of mild degree, and mental symptoms of varying intensity. Associated with these symptoms pericardial friction rubs over the base or the entire cardiac area may be heard. They are at times altogether absent (Cases I. and II.); then follows the rapidly increasing, pear-shaped precordial area of dulness extending upward to the left second rib, to the right

in the fifth, fourth, and third intercostal spaces, at times reaching the right midclavicular line, and to the left to and beyond the left midclavicular line. It is important to note that the apex beat, if present, is within this area of dulness and not at its extreme limits. The shape of this dull area varies with the amount of fluid in the sac. If the quantity is not large the dull area is pear-shaped with its base exaggerated. If the quantity is large the dulness is almost circular or ovoid at its upper border, the centre of the bow being the sternum. The lower dulness merges with that of the liver or even the spleen, but more often with the dulness or flatness of the consolidated lung or that produced by fluid in the pleura. The apex is either lifted to the fourth place or disappears altogether, or the entire heart is bodily displaced to the right or left, according to circumstances.

The sounds at the apex grow distant and faint, and at times can be heard with difficulty. The left axilla presents a hyperresonant note with exaggerated breathing; the left base may show a spot of compression dulness with bronchial breathing. To clinch the diagnosis an exploratory puncture should be made in that portion of the dull area farthest away from the ascertained position of the heart, preferably in the fourth or fifth space to the left of the sternum.

Differential Diagnosis.—1. Serofibrinous pericarditis. 2. Acute dilatation of the heart. 3. Acute catarrhal gastritis. 4. Right-sided or left-sided pleural effusion or empyema, either free or loculated. Some unusual conditions have been mistaken for pericarditis, such as mediastinal tumor, acute endocarditis, cases of aortic regurgitation, and we have recently heard that perforation of the bowel was diagnosed by two very competent observers, the man's condition, however, preventing operation, and the autopsy showing the presence of a purulent pericarditis.

Before considering any of these points in diagnosis the following anatomical belief usually stated in text-books is questioned: That the heart usually or always, in the presence of large quantities of fluid in the pericardium, is pushed away from the chest wall. with the consequent disappearance of the apex beat and weakness of sounds. The heart is fixed at its base by the attachment of its vessels, and this leaves the apex and the ventricles as the movable portion to be effected by pressure from all sides or any side; if,

however, adhesions already exist between the pericardium and the heart on either the right or left side a lateral dislocation of the heart will occur; or pressure exerted upon the filled pericardium as a whole may cause a lateral dislocation of the heart; perhaps other unknown conditions within the pericardium may produce the same lateral displacement. We hope in the future to be able, with the kind assistance of Dr. Longcope, to experimentally verify some of these views; as yet we have not been able to do so.

1. Serofibrinous pericarditis is usually the result of rheumatic infection or concurrent with tuberculosis or Bright's disease. The history is of decided importance as the causative factor. The symptoms and signs are, of course, much the same as in purulent pericarditis. You have not, however, the usual chest complications seen in the purulent variety. The temperature is apt to be but slight, and the leukocytosis does not reach such a grade as in the purulent form. This fact, however, is not to be relied upon. Cardiac dilatation is common in the rheumatic pericarditis, and endocarditis frequent. Dilatation of the heart is not common in purulent pericarditis. The physical signs in both conditions are similar except that we must recall the fact that purulent pericarditis is seldom present without a purulent pleurisy, a pneumonia, or meningitis, or pus or ulceration near the pericardium. Exploratory puncture settles the doubt at once.

2. Acute dilatation of the heart is a common feature of chronic valvular disease and rheumatic pericarditis, but according to Still is uncommon in purulent pericarditis. It is attended with weak, rapid, and irregular pulse. The cardiac impulse, however, is visible, wavy, and occupies several interspaces, although at times it is not palpable; the impulse may be irregular, now weak and now strong, and can be perceived in the epigastric region. The heart sounds are heard, the first short, sharp, but weak, and not either distant or absent. There is a decided increase in the area of cardiac dulness, which, however, does not extend so high as in pyopericarditis. The second sound is sometimes split with so-called "gallop rhythm." Careful mapping out of the heart dulness shows the mobility of the heart. In dilatation there is a tendency to venous congestion and dropsy; the liver increases in size, the kidneys fail to secrete, and

the lungs fill up. The x-ray examination is a decided help, showing in dilatation a fixed quadrangular shadow; in pyopericarditis a movable upper line of dulness. The blood count will show the absence of leukocytosis in dilatation. When dilatation of the heart occurs in rheumatic pericarditis in a case in which friction rubs have been easily demonstrable, the diagnosis becomes extremely difficult. It rests mainly on the points mentioned above, yet we have seen the diagnosis of acute pericardial effusion with request for immediate opening of the pericardium made by a most able diagnostician, and the operation show an acute cardiac dilatation.

3. Acute catarrhal gastritis presents the history of some causative factor, such as the ingestion of indigestible food, drink, or irritating substance. The vomiting is more or less constant and the pain confined to the gastric area. The temperature is usually high, and cough and dyspnœa are not present. The cardiac outline is normal in size and the heart sounds well heard. The blood is of little help, as a leukocytosis may be present in acute gastritis.

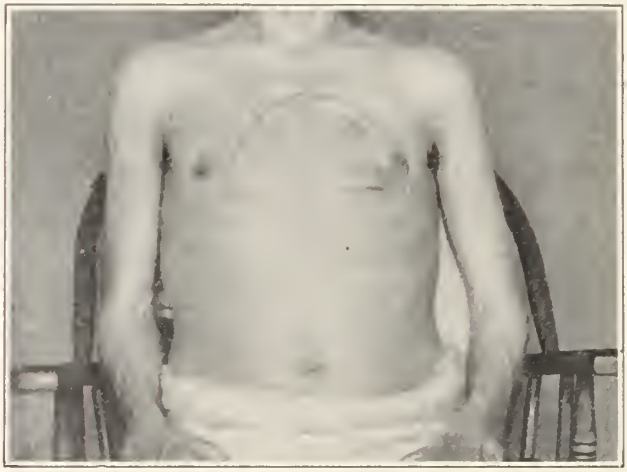
4. Pleural effusion, either free or loculated. It is taken for granted that the reader is familiar with the signs of free fluid, be it either serum or pus, in the chest. When this is present in sufficient quantity so that the lower limit of dulness reaches the anterolateral portions of the chest and impinges upon the normal heart dulness, we have a condition which presents many difficulties in diagnosis. If the case has been seen from the start and the size of the heart and the character of its sounds carefully noted, the diagnosis is perhaps not difficult, but when brought face to face for the first time with the presence of large quantities of fluid in the chest, with the dislocation of the heart either to the right or to the left, and apparent increase in the outlines of the pericardial sac, the diagnosis becomes extremely difficult. Under such circumstances the liberation by either aspiration of the serum or resection of a rib and thorough drainage of the pus is, we think, essential to an opinion. With the relief of the fluid the heart will return toward its normal site. If there remain an unusual area of precordial dulness there is a great probability that the pericardium is also filled. The great frequency of involvement of both the pleura and pericardium in pneumonic cases must be remembered. Of course, change in posi-

tion of the patient will in large pleural effusion or empyema alter the upper line of dulness. This will, however, give you but little information with regard to the pericardium. Loculated empyema is usually latent and extremely difficult to localize. Unless large it is not apt to dislocate the heart except when present in the antero-lateral part of the chest. When so situated the heart is dislocated to the right or left, and a visible impulse and apex beat can still be seen. The symptoms of a loculated empyema, cough, fever, and sweats, with the consequent leukocytosis persist. The pulse remains good, the heart sounds are readily heard and are not weak. There is no evidence of pericardial friction.

Case I. (Reported in *New York and Philadelphia Medical Journal*, January 30, 1904.)—An Italian laborer admitted to hospital on the sixth day of pneumonia of the right base. Consolidation of left base on ninth day, with an extensive pleurisy. Pericarditis diagnosed on the thirteenth day. Paracentesis performed with immediate relief. Reaccumulation and formal operation on January 31st under local anæsthesia. Slow but complete convalescence at the end of six weeks' time.

M. D., an Italian laborer, aged thirty-six years (Hospital No. 3215) admitted to the Pennsylvania Hospital on January 4, 1903, with history of six days' illness, beginning with a chill, cough, and pain in the right side. On admission physical signs of consolidation of the right base were present. From the start the pulse was weak, and heart sounds feeble, though the heart was not displaced and cardiac outline normal. Respirations were rapid (48 to 56), and the leukocytes (ninth day) 8800. On the ninth day the base of the left lung showed signs of consolidation with extensive pleurisy, which was especially marked in the left mammary region. The temperature for the first four days ranged between 103° and 102° F., and gradually fell by the twelfth day to 100° F., in the morning, with an evening rise of a degree or more. This low temperature was maintained from this time on. The right base cleared slowly. By the thirteenth day the patient appeared very sick, with grayish pallor, dyspnœa and weak, rapid pulse. No pericardial friction was heard, but the pericardium was considered involved by the thirteenth day, empyema of the right base having been excluded by puncture. The apex beat now was neither visible nor palpable, and there was impaired resonance in the fifth and fourth interspaces to the right of sternum. This area of cardiac dulness increased, especially on the right side. The cardiac sounds were very indistinct. The epigastric region was full and very tender. The paradoxical pulse was marked on the eighteenth day of illness, January 16th. The leukocytes, before not increased, had now risen to 18,600. An exploring needle

inserted in the fourth right intercostal space showed the presence of light-greenish, turbid fluid, which on culture showed a pure pneumococcic growth. The pericardium was aspirated at the same spot the following day and 420 c.c. of seropurulent fluid were withdrawn, with great relief to the more urgent symptoms. In a week's time rapid reaccumulation occurred, and, as was expected, exploratory puncture now showed thick, yellowish pus in the pericardium. The patient refused operation primarily. Permission to drain the pericardium having finally been secured, Dr. Le Conte operated on January 31st. Chloroform anæsthesia was at first attempted, but resulted in such struggling and cyanosis that local anæsthesia with cocaine was at once substituted and the fifth space incised at the site of the



CASE I.—The upper line shows the outline of dulness when the pericardium was filled with purulent fluid; the smaller outline of cardiac dulness six weeks after operation.

normal apex beat. The pericardium was readily opened and between a pint and a quart of non-odorous pus slowly escaped. The laboratory examination of the fluid showed many leukocytes and pneumococci.

The man was very ill for two weeks after operation, with weak, irregular pulse and but slight fever. From February 22d to 27th there was slight œdema of the face, back, and ankles present, though the urine during this time was free from casts, only leukocytes being present. At no time subsequent to the operation were pericardial friction sounds heard. The drainage-tube was removed in twenty-six days and the patient was permitted to leave the hospital on February 23d, fifty-one days after operation.

It is interesting to note in this case the very mild degree of fever present while the pericardium was filled with seropurulent and purulent material.

This patient was seen December 31, 1903, almost a year after operation. He looks well and has gained flesh, and has been doing light work in a store. He states that he is sleepy a good deal of the time and has, on deep inspiration, some pain over the heart and in the back. No dyspnœa or cough is present. Appetite and digestion are good. Pulse is 76, small in volume, and very compressible. There is no œdema of feet. Liver is normal in size.

Inspection of Chest. No venous phenomena of neck; no retraction of interspaces over the cardiac area or behind (Broadbent's sign). The outline of the cardiac dulness is normal. The apex in the erect position is in the fifth interspace within the midclavicular line. It is definitely movable with change in position. When he lies upon the right side the apex falls at least an inch toward the sternum, and lying on the left side pulsation is visible and palpable one inch outside of the nipple.

Auscultation. First sound is rather short, somewhat liquid, and with weak muscular tone. Second sound is clear; pulmonic sounds are distinct and normal, while the aortic sounds are rather weak and clear.

The features of interest in this case are:

1. The absence of leukocytosis during the pneumonia and its development with the pericarditis.

2. The absence of the pericardial friction sounds, both before and after operation.

3. The absence of fever, subnormal temperature with pus in pericardium.

4. Recovery without the physical signs of an adhesive pericarditis.

Case II.—This case is one of lobar pneumonia complicated with empyema resulting in operation, with death eight days after operation; pyopericarditis overlooked.

The man, S. J. B., aged fifty-two years, was admitted to the Pennsylvania Hospital on December 21, 1903 (No. 2860). His family history is unimportant. Except for a pleurisy four years ago, which lasted three months, has had no serious illness. No venereal history. Formerly drank excessively, but periodically. Smokes freely. Is an attendant for feeble-minded children in Vineland.

Eight days before admission to the hospital he had a severe chill, which was followed by pain in the right side. He had a slight cough with a little whitish expectoration. On admission he presented unusually small but equal pupils, urinous breath, with dry, parched tongue. Both the apex and base of the right lung are consolidated; the middle lobe escapes. The liver is increased in size. The abdomen is large; distention marked. The heart is either dislocated or increased in size, the right border being the right edge of the sternum, the left border anterior axillary line, the upper

border the second interspace. The sounds are heard distinctly, but are not strong. The apex beat is palpable two inches outside the midclavicular line in the fifth space. No murmurs are present. The temperature on admission and for the first forty-eight hours was about 102° F. On the ninth day there was a fall to 100° F., with subsequent rise (pseudocrisis), and on the eleventh day to 99° F. From this time the temperature is but slightly elevated, about 100° F. The most extreme tympanites developed, which, it is interesting to note, was relieved by eserine. By the twentieth day of the sickness, or twelve days after admission, the signs at the right base indicated fluid. At this time, however, the leukocytes had fallen from 22,700 on the fourteenth day to 12,600. Exploratory puncture on February 5th showed the presence of pus. At this time it was noted that there was impaired resonance in the third and fourth interspaces to the right of the sternum. It was, however, supposed to be due to the line of fluid. The fifth rib in midaxillary line was resected by Dr. Le Conte and showed the presence of a large quantity of thick, yellowish pus in the right pleural sac. He did not do well after the operation, the pulse growing weaker and dyspnœa increasing; the temperature remained low. I regret to say that I did not see him after he was operated upon.

The autopsy showed, in addition to an unresolved pneumonia of the upper and portions of the middle and lower lobe, that the right pleural cavity was well drained, but that a purulent exudate filling the pericardium had been entirely overlooked. The pericardium contained 600 c.c. of thick, yellowish pus.

Case III.—Croupous pneumonia of the left base with the development of pericardial friction on the sixth day of illness; partial disappearance of the friction, with disappearance of the apex beat and great increase in the area of cardiac dulness. No evidence of fluid at the base at this time; incision into the pericardium, evacuation of pus; second operation in eleven days after the first, with drainage of some pus from the left pleura; rapid convalescence and recovery.

D. S. (No. 3141), aged thirty-three years, an Italian laborer, admitted to the Pennsylvania Hospital on January 14, 1904, complaining of pain in the left chest and back, with cough and dyspnœa, and fever of twenty-four hours' duration. On the second day it was evident he had consolidation of the lung involving the left base, faint bronchial breathing being heard at the extreme base and in the anterior axillary line. From the start the pulse was rapid, weak, and compressible. The man was very ill, with active delirium. Complained much of pain, especially in the epigastric region and around the left costal margin. On admission the heart dulness was normal in size, no friction sounds were heard, and the heart sounds at the apex were very weak. On January 18th, or the sixth day of the illness, pericardial friction sounds were heard in the left third, fourth, and fifth intercostal spaces. The apex was not visible but palpable in the fourth

interspace. By this time bronchial breathing was readily heard all over the left base. On the ninth and tenth days friction sounds were heard at the apex and to the right of the sternum; by the twelfth day there was dulness in the interspaces to the right of sternum, with very weak pulse, dulness, and absent tactile fremitus in the left axilla. There was no evidence of fluid posteriorly. The leukocytes on the third day of the illness were 17,900; on the seventh day, 16,400; on the tenth day, or the period during which pericardial friction and increase in the area of cardiac dulness occurred, 28,000.

The temperature ran up to 103° F. until the seventh day, when a pseudo-crisis occurred, with a fall to 100° F., subsequent rise and fall, and on the ninth day to 99° F. The pericardium was considered filled on the twelfth day. An exploring needle was introduced in the fifth interspace one inch within midclavicular line, and purulent fluid readily secured. Operation was performed within one-half hour under Schleich solution by Dr. Le Conte.

About a quart of pus welled through the incision in the fifth interspace. Upon sudden paroxysmal coughing a large quantity of pus, estimated two quarts, was suddenly expelled from the chest through the incision. It seemed as though an empyema must have ruptured through the incision. It is estimated that three quarts of pus were removed, which showed on culture pure pneumococcus growth. For eleven days the temperature remained between 100° F. in the morning and 102° F. in the evening, with rather rapid pulse (112 to 116); respiration about 32.

Unfortunately, the wound was permitted to close up about six or seven days after this operation. The case about this time was transferred to Dr. Harte's care, and after consultation with him it was considered advisable to reopen the chest to establish drainage, this time into the pleura, with the result that a small quantity of pus was recovered. This was done on February 6th and the twenty-fifth day of the illness. From this time on his convalescence was rapid and complete.

This case has been the subject of considerable discussion among his physicians; it has been questioned if the primary operation opened the pericardial sac, and the opinion expressed by those who did not see the case medically was that we are here dealing with an empyema and not pyopericarditis; that is, a loculated empyema was present with dislocation of the heart to the right side, and that the operation opened up the pleural sac and not the pericardium.

Case IV. (Seen in consultation with Dr. Richard H. Harte in the surgical wards of the Pennsylvania Hospital.)—N. M., an Italian laborer, aged twenty-two years. Both of his feet were frozen, and he subsequently developed moist gangrene. Operation (amputation of both feet at lower

third leg) after twenty-four days. Profoundly septic at this time. Sloughing of flaps, with granulation. Tardy convalescence. From the middle of February and through March the development of temperature with extensive increase in the precordial area of dulness; lungs clear. Operation, with drainage of the pericardium, on March 14th, with gradual convalescence.

N. M., aged twenty-two years, laborer, admitted to the Pennsylvania Hospital on December 21, 1903. During the cold weather of December this man slept in a barn, in which he was found on the day of admission with both feet frozen. On admission they were perfectly cold and of slate color to about four inches above the ankle. In twenty-four hours blebs formed over both heels, with return of normal color to the skin above the ankles. Moist gangrene developed in several days' time. By January 10th the lines of demarcation present over both insteps with the development of septic temperature. He refused operation, but finally double amputation was consented to and done on January 14th. The flaps, however, did not do well, sloughed and healed slowly by granulation. By January 29th the temperature reached and remained normal for some time. During February, however, there was a gradual increase in temperature without any unfavorable signs. Pulse and respiration increased in frequency, with sweats at intervals. No pain or cough or chill. It is noted, however, that the heart is dislocated to the right, and visible pulsation is present in the third, fourth, and fifth interspaces, and the apex of the heart seems to be almost at the right nipple. On March 3d, examination shows that the cardiac dulness extends one inch to the right of sternum. The heart's action is embryonal. There is no cough or expectoration. On March 13th Dr. Harte asked me to see this case with him. There was present a large circular area of dulness over the precordia, extending one and a half to two inches to the right of sternum, to the second rib and outside the left midclavicular line. No visible pulsation is present. The heart sounds are loudest to the right of sternum. No friction is present. Paradoxical pulse not present. No venous phenomena in neck. The base and axillary region of left lung are clear. A needle inserted into the fifth interspace within the midclavicular line secures light-yellow, purulent fluid, which on examination shows the presence of streptococci. The following day Dr. Harte resected the fourth and fifth ribs within the nipple and secured a large quantity of pus. The man has slowly convalesced, but the heart has not returned to its normal position. There is still visible pulsation in the third right space, while the apex seems to be in fourth space (adherent pericardium?). No Broadbent sign.

The points of interest in this case are:

1. The hæmatogenous or embolic origin of the pericarditis.
2. The unusual delimitation of the pericardial outline.

3. The absence of cough and pain.

4. The differential diagnosis between purulent pericarditis and dislocation of the heart by loculated empyema.

SURGICAL CONSIDERATIONS, BY ROBERT G. LE CONTE, M.D.

In presenting the surgical side of pyothorax the limited time at our disposal will prevent a discussion of the subject in full, and we therefore propose to deal only with three questions of importance to the surgeon: 1st, exploratory puncture for diagnostic purposes; 2d, anæsthesia; 3d, intercostal incision or excision of costal cartilages.

1. Exploratory Puncture. Purulent pericarditis cannot be definitely diagnosed unless the fluid is in hand for inspection. For this reason exploratory puncture is an essential procedure in making the diagnosis. As we cannot tell the presence of pus by the degree of severity of the symptoms, the feeble pulse and cardiac action, etc., a formal operation for opening the pericardium should not be undertaken until pus has been proved present. One drop is sufficient for a diagnosis, and this can usually be obtained with a slender needle similar to the ordinary hypodermic needle, but somewhat longer.

The three principal dangers in exploratory puncture are wounding the heart, opening a large bloodvessel, and infecting the pleura if septic material be encountered in the pericardium.

A wound of the heart is grave according to its size and depth; a small wound need not of necessity cause any symptoms. The cavity of the ventricles has been aspirated and life saved in acute dilatation of the heart, and it is a recognized and legitimate surgical procedure. If, therefore, a small needle is used no serious complication need result if the heart is punctured. When the heart is adherent to the anterior surface of the pericardium, or when the diagnosis of pericardial distention is at fault, there is great probability that the heart muscle will be penetrated by the needle. Should the needle puncture the heart it would readily be recognized by the motion imparted to it.

For the same reason the dangers resulting from the wounding of a large bloodvessel are insignificant, and need not be regarded if our

needle is small. We can readily avoid the usual position of the internal mammary vessels, and no other vessel need be feared except in the presence of some anomalous distribution.

To appreciate properly the dangers of invading the pleural cavity during paracentesis we must turn for a moment to the anatomy of the part. The anterior mediastinal space, equivalent to that portion of the pericardium which is not covered by the pleuræ, practically commences at the level of the upper border of the fourth costal cartilage and extends downward to the diaphragm. The left pleura recedes much farther from the median line than the right, so that usually the edges of the left pleura do not reach the border of the sternum. For the purpose of obtaining some definite knowledge on the width of the anterior mediastinal space, 11 observations were made on the post-mortem table, and we are indebted to Dr. W. T. Longcope, the pathologist of the Pennsylvania Hospital, for the careful measurements and notes of these cases. From these observations we judge that the average width of the anterior mediastinal space, on a level with the fifth costal cartilage, is from 4 to 5 cm.; but that the space is quite variable is shown by the measurements, in one case being 8 cm. and in another 1 cm. In the latter case, with a width of 1 cm., the edge of the left pleura just reached the border of the sternum. In two other cases, where the widths were 3 and 4 cm., respectively, the left pleura reached nearly to the edge of the sternum. In the remaining cases the border of the pleura was some distance from the sternum. The right pleura in every instance reached beneath the sternum.

In this region the left pleura is rather firmly attached to the triangularis sterni muscle, but can easily be dissected from it. Its attachment to the pericardium is a little more firm, but in a normal subject it can readily be separated until it dips over the side of the pericardial sac. For some distance, then, dependent upon the position assumed by the pericardium in the left chest, these two layers of parietal pleura are in close apposition. The substance of the membrane is so thin, frail, and translucent that it would be hard to conceive of any inflammation which involved the pericardium that would not also extend to both layers of the overlying pleura and obliterate the pleural space in this region.

The pericardium, although it does not contain elastic fibres, is capable of very great distention, and its cavity will hold quite two litres (Osler). The lungs are displaced and may be thrust far backward and outward in the presence of such distention, but the pleuræ, on account of their attachments, keep their usual relations to the anterior thoracic wall. That pathological lesions change the pleural relations of the pericardium very little is shown by the dissections of Delorme and Mignon[1] in cases of pericarditis and in bodies where fluid was injected into the pericardium. We cannot compare distention of the bladder and its concomitant displacement of the parietal peritoneum with distention of the pericardium, for the peritoneum in the region of the bladder is very loosely attached to the anterior abdominal wall, while the pleural attachment is much more firm to the triangularis sterni muscle, and still more firm to the pericardium. We therefore agree with Roberts in believing that Fowler is in error when he states that the boundaries of the pleuræ are greatly displaced in pericardial distention. As the pericardium probably distends equally in all directions, the anterior mediastinal space must be broadened to some degree by this portion of the pericardium distending proportionately with the remainder of the walls of the sac. The more the pericardium is distended the greater will be the surface of pleural contact, and when this surface adheres the larger will be the operative field where the pleura can be crossed with safety.

To return to the observations on the cadaver, pins were thrust through the fourth and fifth interspaces to the right and left of the sternum, hugging the border of the sternum as closely as possible. It was found that on the left side the pins traversed the cavity of the pleura in both the fourth and fifth interspaces in one subject in which the breadth of the anterior mediastinal space was 1 cm.; that in two other subjects, in which the anterior mediastinal space was respectively 3 and 4 cm. broad, the pins in the fourth and fifth left interspaces grazed the pleura but did not penetrate its cavity. In the other eight cases the pins entered the pericardium some distance from the pleural border. On the right side, in every instance, the pins passed through the pleura before reaching the pericardium.

[1] Revue de chirurg., December, 1895, p. 830.

With such anatomical data, we conclude that the point of election for paracentesis, or exploratory puncture, is in the fourth or fifth left interspace, as close to the edge of the sternum as the needle can be made to enter. In this position there is the smallest chance of penetrating the pleura, and the internal mammary vessels should lie well to the outer side of the needle. If exploratory puncture is practised in the fifth right interspace, the point suggested by Rotch, the pleura will almost invariably be traversed. Puncture in the space to the left of the xiphoid cartilage necessitates passing through the diaphragm, and therefore dangerously close to the peritoneum.

The ordinary hypodermic syringe, armed with a needle an inch and a quarter or an inch and a half long, is introduced in the fifth left interspace. If no fluid is withdrawn, and there is no evidence of penetration of the heart by motion imparted to the needle, the fourth interspace should be tried. If this also fails to demonstrate fluid, the same interspace one and one-half or two inches from the left border of the sternum should be tried. If septic material is not found there can be no danger of infecting the pleura, even though its cavity is invaded. If septic material is present in the pericardium the overlying pleural space is almost certain to be obliterated, and under such circumstances the needle can pass through it without danger of further infection.

2. Anæsthesia. In the presence of a large collection of fluid with feeble heart sounds, the administration of a general anæsthetic becomes very dangerous. In the first case reported this evening the patient after four or five inspirations of chloroform became very cyanosed, with feeble, fluttering pulse, and death seemed imminent. Under such circumstances we are forced to use a local anæsthetic, and our experience with infiltrations of Schleich's fluid has proved most satisfactory. In such cases, where the heart action has not been seriously interfered with, ether would seem to be the best for general anæsthesia.

3. Intercostal Incision and Excision of Costal Cartilages. The choice of operation will be very greatly influenced by the condition of the patient. If a general anæsthetic is contra-indicated, excision of a costal cartilage will be dangerous, for under local anæsthesia it cannot be done without causing considerable pain

and severe pain would be very depressing to the heart. Under such circumstances our choice will be limited to an incision through an intercostal space, or aspiration of a large part of the fluid for the purpose of temporarily relieving the embarrassed heart and permitting later the giving of a general anæsthetic.

Incision is certainly as safe as aspiration with a large needle or trocar, and it frequently permits a recovery without further operative procedure, while aspiration necessitates a later operation. If the pericardium is much distended with pus the overlying pleural cavity is almost certain to be obliterated, and incision through it can be made without risk.

The point of election for incision is the fourth left interspace, beginning one inch from the sternal border and extending to the normal position of the apex beat of the heart, about one inch internal to the anterior mammary line. The tissues are infiltrated with Schleich's fluid and the incision gradually deepened. When the pleura is adherent it cannot be recognized; but if its cavity is free, and the incision has been carefully deepened, air will rush in as soon as it is penetrated and before the pericardium has been opened. When pus is reached it is allowed to flow out slowly for fear of embarrassing the heart by the sudden relief of pressure. A rubber drainage tube is inserted and an ample dressing of gauze applied. This is sufficient for the time being, and the question of flushing the pericardium or resecting the costal cartilages may be put off until the physical signs show that the drainage is not good or that the tube is plugged with membranous lymph.

Intercostal incision produces no shock: it is the simplest procedure in grave cases; it permits a more extensive operation at a later period if found inefficient; it permits the recognition of non-adherent pleural surfaces before the pericardium is opened, and in the majority of cases it will lead to a cure where a recovery is possible from an operative procedure. The question of the recovery of the patient is more dependent upon the cause of the pyothorax and the pathological lesions present in other portions of the body than upon the choice of an operative procedure.

The deductions drawn by Reichard[1] in advocating resection of a

[1] Zur Kasuistik der Operationen bei Pericarditis, Mitteilungen aus den Grenzgebeiten der Medizin und Chirurgie, No. 7, p. 356.

rib instead of incision in the intercostal space, do not seem to us to be justified. He collected 33 cases, in 19 of which incision was practised and in 14 resection of a costal cartilage. Of the 19 cases 6 recovered and 13 died, and of the 14 resections 8 recovered and 6 died. From this statement of figures he concludes that the safest procedure is resection. If, however, we examine these cases more carefully we will see that of the 13 cases dying after incision 1 died on the table, 2 others within twenty-four hours, a fourth while the pericardial cavity was being flushed after operation, a fifth some weeks later following a double empyema operation, and a sixth from joint empyema. These 6 cases would probably just as surely have died after resection, and one is led to believe that perhaps their condition was so desperate that so formal an operation as resection was not used because too prolonged and dangerous. If, then, these 6 failures are subtracted from the 13 deaths, we would have 6 recoveries and 7 deaths following incision. Also, when we examine the cases that recovered under excision, we find that in 1 the disease was primary in the pericardium, and that 2 others were the result of stab wounds—in a sense, therefore, primary also. Such favorable cases would probably have recovered as rapidly with simple incision, and, if so, by removing them from the tables of the recoveries we would have 5 recoveries with 6 deaths. In other words, the statistics of the two procedures may be regarded as very nearly equal in the same class of cases.

If excision becomes necessary we are inclined to think that the chondroplastic flap suggested by Roberts is better than the removal of one or two costal cartilages. Roberts cuts his flap through the fourth and fifth costal cartilages, and turns it upward, using the soft tissues of the third interspace as a hinge. On the normal cadaver it gives a good exposure of the internal mammary vessels and readily permits their ligation if they cross the field of operation, and the border of the left pleura is easily recognized and can readily be dissected free and pushed aside if its surfaces are not adherent.

Conclusions. 1. Purulent pericarditis is quite frequently overlooked in lobar pneumonia and in other pyæmic states.

2. Its presence modifies the pre-existing disease to a considerable extent.

3. In diseases with high temperature the presence of purulent pericarditis depresses the fever range and increases the respiratory and pulse rate without sufficient clinical evidence of trouble elsewhere than in the pericardium.

4. It is yet to be proved that the heart, in case effusion is present in the pericardium, is always dislocated backward and upward. It is probable that in some cases the presence of fluid in the pericardium dislocates the heart either to the right or to the left. (This, of course, will occur if previous adhesion between the heart and the pericardium has occurred on either the right or the left side.)

5. The diagnosis once made the only treatment should be incision and thorough drainage. (Paracentesis will at times relieve temporary urgent symptoms and should be performed without the slightest fear.)

6. That exploratory puncture can be made with safety and is essential for diagnostic purposes before operation is undertaken.

7. That such puncture should be made in the fourth or fifth left intercostal space, as close to the sternum as possible, with a fine needle.

8. That pericardial dilatation does not alter the relations of the pleuræ to the anterior thoracic wall.

9. In the presence of pyopericarditis the overlying pleural space is usually obliterated by extension of the inflammation.

10. That incision under local anæsthesia is the preferable operation in the majority of cases.

11. That Roberts' chondroplastic flap is preferable to an ordinary excision of costal cartilages when incision fails to give adequate drainage.

PERICARDITIS: ITS SYMPTOMS AND DIAGNOSIS.[1]

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PERICARDITIS is, as a rule, more damaging to the heart than endocarditis. It is almost certain to shorten the life of the person afflicted unless an intercurrent disease should anticipate its effects.

Its causes, to which, merely for the sake of completeness, I would briefly refer, are in the main twofold: direct extension and hæmatogenous infection. Traumatic pericarditis is so rare that it may be disregarded.

The pericardium may be involved by direct extension from neighboring structures in the thorax, such as the lung, the pleura, the mediastinal and bronchial lymph glands, and the œsophagus; also from the endocardium or the myocardium, from the great vessels near their origin, and from aneurysms. Inflammatory processes below the diaphragm may likewise cause pericarditis by extension.

Hæmatogenous infection is the most frequent, and gives rise to the type of pericarditis accompanying acute articular rheumatism and other infectious diseases. A form of particular interest is that associated with Bright's disease and uræmia, the true nature of which is still in doubt. Unquestionably, however, its cause is hæmatogenous, and is either a toxin or a bacterium. It may develop in any of the forms of nephritis, but is most frequently encountered in acute Bright's disease, in the early stages of the large white kidney, and in chronic interstitial nephritis. Frederick T. Roberts says that it is almost never found in cases of typical large white

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kidney. In one of my cases, however, the kidneys were in an advanced stage of chronic parenchymatous nephritis (the so-called large white kidney); but, as the patient had pneumonia also, the share of the kidneys in producing the pericarditic process is not readily fixed.

Nephritic, or uræmic, pericarditis is nearly always of the fibrinous variety, with little fluid; but it may be serous or hemorrhagic. I am not forgetting simple hydropericardium, which is sometimes present in cases of chronic nephritis complicated with general dropsy and effusions into the serous cavities. I have, however, been surprised at the rarity with which hydropericardium occurs when the conditions supposed to favor it, renal and cardiac dropsy, are present.

Pericarditis is quite common in cases of pneumonia, especially when this disease is on the left side. It is frequently overlooked, but should always be thought of as one of the possible causes of so-called delayed resolution, or, rather, delayed crisis. In my experience it has usually been purulent; but it may be serofibrinous. It is generally accompanied with a mediastinopericarditis or a pleuropericarditis of the same nature.

I must say a few words in regard to pericarditis of tuberculous origin. This is usually of the plastic, obliterating variety, but sometimes it is purulent. It is rare, and is commonly overlooked during life; and even after death its true nature may not be recognizable without a microscopic examination.

In taking up now the symptoms and diagnosis I shall not discuss these two aspects of the subject separately, but shall consider the principal features as they present themselves to the examiner.

1. The Friction Sound. This is most marked in acute fibrinous pericarditis, the commonest form. Its site of maximum intensity is to the left of the sternum in the third and fourth interspaces, and sometimes in the second. In rare instances, as in two under my observation at the present time, it is heard first or solely at the apex. It is usually of a churning character, superficial, and markedly intensified by pressure with the stethoscope. It may have a gallop-rhythm, and in pronounced cases it has a peculiar chugging quality, like the noise made by a locomotive just starting to move.

If confined to the apex it may closely resemble a presystolic murmur; while when loudest at the base it may simulate double aortic disease. The character of the pulse, the size of the heart, and the history, together with careful examination so as to discover the second aortic sound, will aid in the diagnosis from aortic-valve disease.

When the friction sound is loud it is often difficult to distinguish existing endocardial murmurs. These may be heard if a double stethoscope is used and no pressure is applied. Pressure brings out the friction sound and thereby renders the murmurs faint or inaudible. I have recently been able to study the heart before the development of pericarditis in two cases. One was that of a young Italian with aortic regurgitation. In the other case, also that of an Italian, there was mitral insufficiency. As the friction sound increased in loudness the murmurs in both grew fainter and fainter, and at present the aortic murmur is inaudible; while the mitral systolic can be heard when the stethoscope is placed lightly upon the chest. In some cases a mitral murmur, obscured in the precordial area by the confused noise of the friction sound, may be heard in the axilla.

Pleural and pleuropericardial friction must also be differentiated from the ordinary pericardial friction. Both the former are modified by respiration.

The existence of a friction sound does not exclude the presence of fluid. In one of the cases in which Dr. Roberts tapped and obtained considerable fluid, the friction sound had not disappeared. The fluid is often held in compartments separated from one another by bands or areas of adhesion which preserve the friction sound. In some cases the friction sound persists at the apex after it has disappeared elsewhere, probably because the greater movability of the apex retards the formation of adhesions.

Friction may cause a palpable thrill. Of my recently observed cases this thrill was present in two out of nine. The loudest friction may, however, be entirely unaccompanied with thrill.

2. Pain and Tenderness. These were present in four of the nine cases. In these four the pain was intense. In three the tenderness was an exquisite hyperæsthesia, so that percussion became torture. In one of the cases the pain was paroxysmal, and was

apparently as severe as in angina pectoris, although the direction of radiation was different. It started at the wrists and ran with great violence up the arm and across the shoulders into the chest. In another case there were moderate pain and tenderness in the epigastrium; and in one severe subjective pain in the præcordia and the epigastrium without any tenderness.

It is not easy to explain why, with no difference in the physical signs, the pain and tenderness should be extreme in some cases and entirely absent in others. It may be that in those with pain there is involvement of the superficial structures, especially the muscles, analogous to that described by Coplin as accompanying pleurisy and pneumonia.

3. The Apex Beat. In the fibrinous form the apex impulse is diffuse, indistinct, and sometimes undulatory, and can usually be appreciated better by touch than by sight. In effusions of any size, it is commonly absent; but a more or less vigorous apex beat is entirely compatible with the presence of a large effusion, as was strikingly shown in a patient that came under my observation at the Polyclinic Hospital. Even when the apex beat cannot be seen, it may often be felt as a distinct shock—a point of importance in differentiating between dilatation and effusion. When the apex beat can be located it is found above the lower level of dulness.

4. Pericardial Effusion. Few conditions are more frequently overlooked than effusion into the pericardial sac. At times the signs are unmistakable, even upon superficial examination; at other times strong doubt encompasses the diagnosis. The signs to which value should be attached are:

1. The character of the right border of the heart, which becomes convex.

2. A change in the cardiohepatic angle, which, from having been a right angle, becomes an obtuse one.

3. As a corollary, the presence of Rotch's sign, dulness in the right fifth interspace, close to the sternum.

4. An increase in dulness upward as far as the second interspace.

5. Flatness with intense resistance on percussion.

6. The position of the apex beat with relation to the lower boundary of the precordial dulness.

7. The shape of the precordial dulness, which is triangular, or, rather, shaped like a trapezium with the base downward.

8. Bamberger's sign, a patch of dulness with bronchial breathing near the angle of the left scapula.

9. Ewart's sign, a separation of the first rib from the clavicle, making it possible to feel the former throughout its length.

10. The presence of fluid on exploratory puncture.

The best place for exploratory puncture and also for aspiration is still unsettled. There is certainly no one point of election suitable for all cases. When the presence of fluid is suspected, and the friction sound is still present over limited areas, it is a good and self-evident rule not to make the puncture where friction is heard, nor should it be made at a point where the heart sounds are very distinct. Another wise maxim is to go in slowly and to arrest the needle as soon as the heart is felt to rub against its point. If the apex can be shown by auscultation—or, perhaps, by palpation—to be above the level of the fluid it will naturally be perfectly safe to tap below it. This will usually bring the point of puncture in the sixth interspace, about the nipple line. At other times the fourth or fifth interspace on the right may, with advantage, be selected.

The presence of fluid may be simulated by dilatation, and differentiation of the two conditions is far more difficult than the parallel-column method of text-books indicates. I have mistakenly tapped in a case of this kind, and others have had a similar experience.

The position of the apex beat with relation to the lower boundary of the heart is of value in the diagnosis; also the presence of a decided cardiac impulse, especially a pronounced diastolic shock. Search should also be made for Bamberger's sign, referred to above.

Great diagnostic difficulties must arise when the fluid occupies an abnormal position; when, for instance, it is collected on the left side of the heart, in which case the precordial dulness extends into the left axilla and simulates pleural effusion. If the fluid is in the pericardial sac, the dulness rarely extends below the eighth rib in the axillary line. Sometimes only tapping can decide the question. When a pleural effusion is co-existent—and this is not at all uncommon—the discovery of the pericardial effusion is rendered far

more difficult. When there is double pleural effusion, the pericardial effusion is usually overlooked.[1]

Sometimes pericardial effusions shift with change of posture, especially when the patient is turned to the right. Fluctuation is said to occur at times, but in the cases that I have seen it was not noticed.[2]

Pericardial effusions sometimes simulate *mediastinal tumor*. This is especially the case when the effusion is of long standing. In both conditions there may be dyspnœa, orthopnœa, engorgement of the jugular veins, intense cyanosis, dysphagia, enlargement of the veins of the chest, and extreme flatness on percussion. As at times there is no history suggesting an antecedent pericarditis, suspicion of a mediastinal tumor in these circumstances becomes not unreasonable. A year or two ago I reported a case of this character to the Section in Medicine of the College of Physicians. The patient had the signs that I have mentioned. The cyanosis and dyspnœa were very marked, and the flatness on percussion was most striking. The onset had been insidious, and there was no history of any previous illness. The man was not bedfast, but was able to walk to the Polyclinic Hospital on the clinic days. I took the view that the case was one of either pericardial effusion or mediastinal tumor, although the latter seemed the more likely; but the man died a few weeks later of exhaustion, and the autopsy showed hæmopericardium, the origin of which was not revealed. It is a curious fact that in another case in which the diagnosis of mediastinal growth was for a time entertained, hæmopericardium was found.

Pericarditis with effusion sometimes simulates *pneumonia*. The distended sac compresses the left lung; an examination of the latter may show impaired resonance and a patch of bronchial breathing in the back. The existence of fever, cough, leukocytosis, dyspnœa, and cyanosis strengthens the suspicion of pneumonia. In a case of this kind that came under my observation at the Philadelphia Hospital, the cyanosis and general prostration were out of proportion

[1] In one of Thayer's cases, just published (Johns Hopkins Medical Bulletin, May, 1904), the fluid (over 1200 c.c.) was lying posteriorly to the heart and at the lateral angles of the pericardial sac.

[2] In pericardial effusion, just as in hypertrophy of the joint, skodaic tympany may sometimes be detected over the upper part of the left lung.

to the extent of lung apparently involved. Such disproportion may constitute a point of diagnostic significance.

It is well known that morbid conditions in the chest may cause *pain to be referred to the abdomen*, and give rise to the belief that the disease is located below the diaphragm. I need mention only the so-called "abdominal" pneumonia, which is not rare in children and is also seen in adults. This may resemble appendicitis or perforation of the intestine. Less well known is the fact that pericarditis may cause pain of such severity in the abdomen as to simulate peritonitis.

On the other hand, the interesting observations of Ortner (*Wien. klin. Woch.*, June 29, 1899) teach us that in some cases of peritonitis, especially of the hepatic and epigastric regions, a friction sound may be heard over the præcordia in the absence of inflammation of the pericardial sac itself.

A peculiar case, probably belonging to the class described by Ortner, came under my observation at the Polyclinic Hospital in February. A woman, fifty-two years of age, was admitted with severe epigastric pain, tenderness, vomiting, slight jaundice, fever, and leukocytosis. There were no cardiac symptoms, but examination showed the presence of a pronounced friction sound, which was audible from February 3d to February 6th. A few days later a friction sound was discovered in the region of the gall-bladder, extending thence for a short distance upward over the liver. Within a few days this also had disappeared, and the patient left the hospital apparently well.

The only way in which one can escape the error of diagnosing abdominal disease when the trouble is in the chest, and *vice versa*, is by making a careful examination of the thoracic and abdominal organs in all cases.

Even in the early stages of pericarditis signs of *pulmonary congestion* are common, especially on the left side in the lower axillary region. In three of my cases a distinct generalized *bronchitis* was found.

Fever is, as a rule, present in acute pericarditis, but it has nothing distinctive. When the effusion is purulent the fever may be intermittent, even when the quantity of pus is small. The fever in pericarditis may be present for several days before the friction sound is discovered. Until then a case of this kind is usually con-

sidered to be one of tuberculosis, sepsis, or typhoid fever. I have thought that in such circumstances the pericarditis probably begins posteriorly, and that the sudden appearance of the friction sound, several days after the onset of fever is due merely to the fact that the inflammation has extended sufficiently far forward for the friction to become audible.

A curious phenomenon not rarely observed in pericarditis is pronounced *delirium* or other mental disturbance. Da Costa, Balfour, and Musser have made reference to this feature. In one case studied by me with the late Dr. Packard at the Philadelphia Hospital, there was maniacal delirium, which might readily have led astray an observer less thorough than Dr. Packard. Da Costa, in one of his last papers, advised that in every case of acute mania the heart should be examined for pericarditis. The duration of the delirium is variable. In a case of mine, one of tuberculous pericarditis, it extended over a number of weeks and was of a mild character. In his lucid intervals the patient was extremely irritable.

It is difficult to explain the variability in the general reaction of the system to inflammation of the serous membranes. We are accustomed to consider the three serous sacs identical in this respect; but pathology proves them to be different, even if histology does not. The pleural cavity may contain pus for days, weeks, or months without the production of a serious general infection. The peritoneum is a much less efficient guardian of the organism, and suppurative inflammations, as a rule, very speedily influence the condition of the system at large. Acute inflammations of the pericardial sac, whether purulent or serofibrinous, seem to be rapidly productive of constitutional symptoms.

In order to solve these enigmas we must know:

1. The conditions of lymphatic absorption, both as to direction and as to rapidity.

2. The efficiency of the lymph-gland outposts.

3. The richness in nerve supply, upon which the degree of functional disturbance will depend.

Adherent pericardium more often, probably, than any other important condition, comes to the autopsy table undiagnosed. One reason for this is that at times the characteristic signs are altogether

wanting. At others, some or all of them are present but are overlooked. Briefly stated these signs are as follows:

1. Systolic retraction of the intercostal spaces.

2. Retraction of the lower portions of the chest in the lateral and posterior aspects—the so-called Broadbent's sign.

3. Diastolic shock or rebound.

4. Absent respiratory movement in the epigastric triangle. The last three are found especially in cases of mediastinopericarditis.

5. Pulsus paradoxus—the so-called Kussmaul's sign—the pulse becoming weaker during inspiration. This is not of much diagnostic value, except when accompanied by inspiratory swelling of the veins in the neck.

6. Diastolic collapse of the veins of the neck—Friedreich's sign.

7. Marked undulation of the heart beat.

8. Early ascites, preceding œdema of the legs.

9. Marked irritability of temper. I have been struck with this, but cannot say that it is either a constant or an important symptom.

10. In rare cases, mild delirium.

For practical purposes the following statements may be made with regard to adherent pericardium. It may be suspected:

1. In cases in which a friction sound has been heard over a great extent of the præcordia and has disappeared.

2. As a probability, in children having a chronic valvular lesion and marked hypertrophy of the heart. In such cases adherent pericardium is commonly found at autopsy. The extreme stretching of the sac and the consequent pressure of the two layers against each other cause alterations of the endothelium favoring a low-grade infection.

3. In adults and children with chronic valvular disease in whom, with heart sounds of good volume and an impulse of great force, there develop ascites and dropsy of the limbs. In such cases we may infer that there is something more than the mere valvular lesion. The ancillary factor may not always be adherent pericardium; it may be a fibroid myocarditis, but it is often the former.

4. In the cases of so-called quiet ascites, chiefly occurring in young subjects, in which cirrhosis of the liver and tuberculous peritonitis can be excluded. I have reference to the so-called pericar-

ditic pseudocirrhosis, a subject ably discussed before this society by Dr. Kelly.

Since the beginning of the year to the present time, July 11, 1904, 9 cases of acute pericarditis have come under my observation: 2 I saw through the courtesy of my colleague, Dr. Thomas G. Ashton, at the Philadelphia Hospital; 2 occurred in my own wards in that institution; 3 were observed during my term of service at the Polyclinic Hospital; 1 was in a patient of Dr. Francis T. Stewart, and 1 I saw in consultation with Dr. L. Podolski. Of these 9 cases 3 were in the female and 6 in the male sex; 4 of the patients were colored persons; 3 were tapped—1 with a negative result.

Etiologically the cases may be classified as follows: Probably of rheumatic origin, 4; associated with pneumonia and nephritis, 1; secondary to operation for the radical cure of hernia, 1; secondary to gallstone colic, 1; of unknown origin, 3. Of the 9 cases, 7 were associated with endocarditis and 2 were unaccompanied with any evidence of the endocardial lesion; 2 ended fatally, the autopsy showing fibrinous pericarditis with adhesions.

HISTORY OF CASES.

Case I.—I. H., a mulatto woman, single, aged twenty-seven years, admitted to the Polyclinic Hospital on March 8, 1904, with signs of pericarditis. There was a thrill at the base and a pronounced friction sound; lips cyanosed, cough, dyspnœa, respirations between 44 and 68, pulse 150 to 170, temperature 102° to 103°. The patient was hardly able to speak on account of great prostration and sharp pain, which she located over the lower half of the sternum, whence it radiated into the epigastrium and into both hypochondriac regions. The boundaries of the heart were as follows: Right border, a little to the right of the right border of the sternum, slanting outward from the sternal end of the second interspace to a point an inch to the right of the sternum in the fourth interspace, and to the parasternal line in the fifth; upper border, in second interspace; left border, not ascertainable, the dulness extending far into the axillary region.

On March 9th, the day after admission, Dr. Roberts kindly tapped, at my request, and removed three ounces of pale, translucent fluid, clotting almost instantly. The patient was much relieved. The friction sound became louder, and a systolic mitral murmur became audible. Soon, however, signs of pneumonic consolidation appeared in the left axilla, and a

friction sound was heard at the left base. On March 12th Dr. Roberts again tapped, removing four-and-one-half ounces of chocolate-colored, opaque fluid, which did not coagulate on standing, and had an alkaline reaction and a specific gravity of 1035. It contained a large amount of albumin and a great many red blood corpuscles. The urine was highly albuminous, and in the sediment red corpuscles, hyaline casts, and epithelial cells were found. One fresh specimen contained several fibrinous coagula; and another, obtained shortly before death, was somewhat milky, owing to the precipitation of albumins. The fibrinuria and the rapid coagulation of the pericardial fluid obtained at the first tapping led us to conclude that the fibrin factors must be present in the blood in excessive quantities, and that we should probably find an undue amount of clotting in the heart at autopsy—an expectation that was realized. The patient died on March 13th, five days after entering the hospital.

Clinical Diagnosis. Pericarditis with effusion, lobar pneumonia, mitral regurgitation, pleurisy with effusion, and parenchymatous nephritis. The autopsy was made by Dr. B. M. Randolph, to whom I am indebted for a very full report, which cannot be given *in toto.*

Anatomical Diagnosis. Lobar pneumonia, plastic pleurisy, pleurisy with effusion, acute endocarditis, pericarditis with effusion, perisplenitis, parenchymatous nephritis, and fatty liver. The microscopic examination of the heart showed no departure from normal conditions in the musculature. The pericardium was thickened and infiltrated with round cells. The free surface was clothed with a shaggy fibrinous exudate, which included many polymorphonuclear leukocytes and also extravasated blood cells. The staining of sections for bacteria gave negative results. Bacterial cultures were also unsatisfactory on account of contamination.

Case II.—G. W., a negro cook, aged thirty-one years, admitted to the Polyclinic Hospital March 22, 1904. After a brief illness, supposed to be grip, he had been seized with pains in nearly all the large joints. This was soon followed by intense pain in the chest, with evidences of pericarditis.

On admission he complained of pain in the chest and between the shoulder-blades, and of paroxysmal attacks of great severity, beginning at the wrists and shooting up into the shoulders and into the præcordia. Dyspnœa, profuse sweating, pronounced pericardial friction, and a blowing, systolic mitral murmur were present. Dr. Roberts kindly tapped, but only a small quantity of fluid was obtained. Abdominal distention developed, which gradually grew in intensity, leading us to suspect peritonitis. The advisability of operation was considered, but the man's condition was too low to warrant it. He died in a fit of vomiting, March 28, 1904.

The first blood-count showed: hæmoglobin, 66 per cent.; red corpuscles, 3,250,000; whites, 15,800. There was no albuminuria. The second blood-count, March 27th, showed hæmoglobin, 50 per cent.; red corpuscles, 3,090,000; whites, 10,100.

Clinical Diagnosis. Pericarditis, mitral regurgitation, peritonitis.

Anatomical Diagnosis. Acute fibrinous pericarditis, acute pleuritis, acute fibrinous peritonitis, hypostatic pneumonia, hypertrophy of the heart, chronic endocarditis of the mitral valve, catarrhal enteritis, and fecal impaction.

The microscopic examination of the pericardium showed a copious hemorrhagic fibrinous exudate invaded by round cells, and undergoing connective-tissue organization. There was marked fatty change at the junction of the muscular tissue with the pericardium.

Case III.—F. B., an Italian laborer, aged nineteen years, admitted to Dr. Ashton's ward in the Philadelphia Hospital, April 19, 1904, complaining of pain in the ankles and the left arm, with which he had been suffering for ten days. There was a mitral systolic murmur and a to-and-fro pericardial friction. The urine was free from albumin. Red blood corpuscles, 3,500,000; whites, 14,000. The highest temperature was 101°. The pulse was 120 in the beginning, later 70. The respirations were 40 at first, and finally 20. The patient left the hospital June 28, 1904, much improved.

Case IV.—C. B. This is the patient who presented symptoms of peritonitis in the hepatic region with a pericardial friction sound, and whose history has already been given. I would add merely the blood-count: hæmoglobin, 70 per cent.; red blood corpuscles, 3,710,000; whites, 22,200.

Case V.—R. H., a colored boy, aged thirteen years, admitted to my ward at the Philadelphia Hospital April 29, 1904. Three weeks before admission he had pain in the knees and elbows and in the back and chest. On admission there was intense pain and tenderness in the præcordia, shooting down the left arm to the elbow. The patient constantly held his hand over the heart, and expressed his wishes by gestures, being afraid to speak on account of the resulting pain. The cardiac dulness was enlarged. Auscultation was very difficult, owing to the pain caused by the pressure of the stethoscope; but the signs indicated pericarditis and mitral regurgitation. The blood-count was: hæmoglobin, 85 per cent.; red corpuscles, 4,576,000; whites, 11,000. The patient left the hospital on June 20th, much improved.

Case VI.—E. J., aged thirty-three years, a laborer, admitted to Dr. Ashton's ward on April 15, 1904. The clinical data are not recorded. When I examined the patient I found mitral regurgitation and pericardial friction, without any pain in the præcordia. Subsequently empyema developed, for which an operation was done, but the man died. At autopsy the pericardial sac was found obliterated by recent adhesions. The heart was much enlarged, and weighed 960 grams. There was a rigid mitral orifice, with marked stenosis, and dilatation of the tricuspid orifice.

Case VII.—A. S., an Italian laborer, aged twenty-two years, admitted to my wards April 29, 1904. He had been taken ill eight days before admission with cough and with pain in the chest and shoulders. Physical

examination showed signs of bronchitis, a double aortic murmur, and, at the apex, an indistinct presystolic rumbling, and a loud systolic murmur transmitted into the axilla. Four days later a pronounced urticarial eruption with dermographia appeared. On May 12th, for the first time, a pericardial friction was heard. This soon became very intense. Thereafter the precordial dulness increased, the friction sound becoming less distinct. Exploratory puncture yielded no fluid. On May 19th, the patient suddenly became cyanosed and collapsed, and had a severe coughing spell, with blood-tinged expectoration. An immediate venesection of twenty-four ounces greatly relieved him, the pulse dropping from 150 to 90. The venesection was followed by hypodermoclysis of two pints of saline solution. On May 22d there was a sudden return of cyanosis and collapse, with evidences of pulmonary œdema, and another venesection was performed, seventeen ounces being removed. A hypodermoclysis of one pint of salt solution, with half an ounce of adrenalin chloride, was administered. The result was a marked improvement, which has continued. The friction sound has disappeared entirely, and the aortic, diastolic, and mitral systolic murmurs are the only ones now audible. For several weeks the patient has been up and about the ward. A few days ago a severe pain developed in the left loin. Suspecting an infarct in the kidney, I had the urine examined, and blood was found. This had not been present before. A blood-count early in the course of the case showed: hæmoglobin, 65 per cent.; red cells, 4,350,000; white cells, 16,800. A second count of the white cells showed 12,000. The urine in three examinations out of four showed albumin and granular and hyaline casts. As just stated, the last examination showed red blood corpuscles.

Case VIII.—J. S., a male negro cook, aged forty-two years, admitted to the Polyclinic Hospital, in the service of Dr. F. T. Stewart, June 15, 1904, with strangulated hernia. On the second day after operation the temperature rose to 101.2°, and there was a mucopurulent discharge from the wound. Some rales appeared on the chest, and the urine showed a trace of albumin, but no casts. There had been no albumin in a specimen of urine obtained six hours after the operation.

On the thirteenth day I was asked to see the patient, who had had a continuous temperature, supposed to be due to an incipient tuberculosis of the lungs. Examination showed a to-and-fro pericardial friction in the third and fourth interspaces between midsternum and a point one inch to the left of the midclavicular line, without any cardiac murmur. On the eighteenth day an exquisite thrill appeared in the region of the apex, and over this area a very rough friction was heard, the friction sound having disappeared elsewhere over the heart. Both bases of the lungs showed many moist rales, also the left apex anteriorly. Blood-examination gave: hæmoglobin, 60 per cent.; red corpuscles, 3,120,000; white corpuscles, 8900. The patient's condition rapidly improved. The friction sound was

audible for at least ten days, but on the twenty-seventh day it could no longer be heard, and the patient left the hospital in fairly good condition.

CASE IX.—R. B., a girl, aged thirteen years, first seen with Dr. L. Podolski on June 4, 1903, when she had the signs of acute endocarditis following a rheumatic attack. I saw her for the second time on June 28, 1904, when she had pains in her joints, fever, much precordial pain, and marked prostration. The heart was greatly enlarged, with a loud, blowing mitral murmur, but there was no thrill and no friction sound.

I was surprised not to find any friction, as the precordial hyperæsthesia and the attacks of pain in the cardiac region suggested the existence of pericarditis.

On July 9, 1904, I saw the patient for the third time. She then had a very rough pericardial friction without any pain or tenderness in the præcordia, while at the previous examination she would scarcely allow me to touch her chest. I believe that in this and in the preceding case the pericarditis began on the posterior aspect of the heart, and extended forward.

DISCUSSION ON HEART SYMPOSIUM.

DR. HARE: I fear that I will have to confess a rather limited experience in regard to the matters about which we have heard this evening, for none of us have had the opportunity of seeing many of these cases.

With your permission I would like to divide what I have to say into a consideration of certain experiments made upon wounds of the heart and a limited personal experience in regard to wounds of the heart produced in an endeavor to perform paracentesis pericardii. About fifteen years ago I was much impressed, on reading the report of some Italian surgeons in regard to heart wounds, by the statement that a number of the cases which had occurred in Italy had lived for a long period of time after the injury was done to the heart, and that in more than one instance death followed, not from the injury directly, but several hours or days afterward as the result of severe toil. In one particular instance a man, who was stabbed in the heart in attempting to escape from the officers of the law, ran, if I remember rightly, several miles and finally climbed a tree. Some hours after the injury he fell to the ground and immediately died. At autopsy it was found that nature had first closed the opening in the heart by a clot. The man by exertion had increased his arterial tension which had displaced the clot, and he had died from what might be called a consecutive or secondary hemorrhage. This led me to carry out a series of experiments on the lower animals. There are few conditions which can be so exactly compared as wounds of the heart in man and in animals.

In looking over the results of my experiments to-day I was much interested to find an exact reproduction of the case in Italy which I have just quoted, and which also brings us to a point not brought up this evening. The question has been whether recovery can take place without operative interference, and whether it is not possible for operative interference at times to really hasten death. For example, a dog stabbed with a scalpel at four o'clock, at three minutes past four was apparently dead, but was restored to animation by a dash of cold water; at five minutes past four he became conscious, and the respirations were 20 per minute; at eight minutes past four he was walking around the room. This dog became almost perfectly well. Forty-eight hours afterward it was killed with chloroform and an autopsy made. The pleural cavity was filled with four ounces of fluid, and there were two drachms of blood in the pericardium. Examination of heart showed a widely gaping wound of the left ventricle, which was penetrating, and reached to the opposite wall of the ventricle, causing a hemorrhage on the posterior surface of the heart. The posterior wound was not a penetrating one. A clot had formed in the main wound through which blood would not pass. The clot was dissected out, and I have the specimen in my private room at the Jefferson College. This case illustrates the fact that a natural reparative process may actually contraindicate operative interference.

The following points were impressed upon me by my research:

1. That the chief cause of death in stab wounds of the heart is hemorrhage into the pericardium, which causes pressure upon the auricles and interferes mechanically with the action of the heart. If an animal after being stabbed is immediately operated upon the pericardium is found distended and the action of the heart interfered with. If the pericardium is incised so that the clots can escape, the heart almost instantly begins to beat with regularity and ease; whereas, if the pericardium is not opened, or if you introduce fluid into the pericardium you will find the heart is embarrassed. So, I repeat, that the chief cause of death is not damage to the heart, but the mechanical interference with its action.

2. The second cause of death is loss of blood which follows opening of the pericardium where a clot has not blocked the opening.

3. The third cause of death—and this is very important—is a wound involving the coronary artery. This is quite different in effect from the case in which the wound simply punctures the heart-muscle and opens the ventricle, for the pericardium not only fills with blood, but a considerable quantity of blood is prevented from going to the heart muscle. Delirium cordis can be produced by a section of the coronary artery as completely as if that vessel were filled by an embolus. The only other way in which a wound of the heart produces death is by damage to the co-ordinating centre of Kronecher and Schmey. The chances of a stab wound involving this centre are remote.

Recovery occurs by the formation of clot in the opening, and it depends somewhat upon the direction of the incision through the muscle fibres. Certain incisions seem to open cardiac fibres more than others.

Another influence interfering with recovery is caused by the blood clotting around the visceral layer of the pericardium, which interferes with the action of the heart as it tends to contract. In two instances in dogs I found the heart so surrounded by a clot that it was evident that the heart expansion was materially interfered with. It would seem to me, therefore, from my experience, that it is wise to turn out the clot, and that unless the co ordinating centre of Kronecher and Schmey has been damaged or the coronary artery has been cut, the mere wound of the heart is not necessarily followed by death, unless the clot cannot be turned out quickly enough to save the patient's life. I agree with Dr. Gibbon in the necessity for rapidity of operation when the symptoms are urgent.

Another point of interest is that an injury of stab wound of the heart is not as serious to a man as a stab wound of one of the great vessels of the thorax. There are few instances in which a stab wound of the aorta or other great vessels is closed by a clot, because of the pressure and thinness of the wall, whereas the rhythmic contraction and opening of the heart muscle is favorable to closure. When a stab wound of the heart is of any size, the outer lips are often wide apart, but the inner lips close together. The wound is funnel-shaped, and while it may be large externally, the internal portion is rarely large. In the animal I spoke of in which the clot plugged the opening in the heart the wound of entrance was one and one-half inches long, and the inner wound one-quarter inch in diameter.

I would like to say a word or two in regard to cases of pericardial effusion which demand operative interference. I agree with what Dr. Riesman said regarding the difficulty of making a diagnosis. My experience in the last fifteen years has led me to believe that the diagnosis of effusion in the pericardium, when accurately made, is to a large extent a matter of luck. I have seen many instances in which the dilatation with adhesions has given all the signs of effusion, and I have remained doubtful of positive diagnosis before operation or autopsy. A Fellow of this College saw with me in consultation, thirteen years ago, what we thought was a case of pericardial effusion. We attempted to aspirate the pericardial sac in the fifth interspace on the left side of the sternum. When the needle had apparently reached the pericardial sac pure blood was drawn either from the auricle or ventricle. To illustrate how positive we were that pericardial effusion was present, the next day the patient's symptoms still being urgent and the physical signs of pericardial effusion being positive, I attempted a second operation, and a second time punctured a cavity of the heart and drew pure blood. Two experiences of this kind made me timid about paracentesis pericardii. Both operations, however, caused great improvement in the patient's condition. The air hunger of the patient

decreased, and he pronounced himself as feeling better, although I confess that both the other physician and myself felt very much worse. The incident, however, proved that a wound of the heart is not of itself a dangerous thing, unless it happens to strike the coronary artery or the Kronecher-Schmey centre, that has been mentioned.

In trying to perform paracentesis pericardii I again found a great deal of difficulty, and I do not agree with Dr. Le Conte that a fine aspirating needle shall be introduced. My experience is that a fine needle is apt to lacerate the heart muscle and cause hemorrhagic extravasation, and that if it touches a vessel it is apt to make it bleed, and it is difficult to determine when that fine end is in the cavity rather than in the heart muscle, because the heart muscle is not very firm, and if the needle is held firmly the heart will move on the needle rather than the needle on the heart. I would use a moderate-sized aspirating needle with a trocar, and should endeavor to have the point of the needle sharp, but not very long. Many of these needles have a bevelled point almost a quarter of an inch in length; but it should not be more than a sixteenth of an inch—just long enough to push itself through the serous membrane.

Dr. G. G. Davis: I would like to ask whether in these cases the area of dulness from opposite the second costal cartilage in the sternum to the third costal cartilage is increased? Ordinarily the lungs are in contact in the middle line from the second to the fourth ribs, hence we would expect resonance. The pericardium comes up on the aorta, and the lower border of the aorta would be about opposite the angle of Ludwig, which is opposite the second rib.

The question of distention is an interesting one. By experiments upon dead bodies I find it impossible to produce what I would call a very satisfactory specimen of pericardial effusion, for the reason that the distention must be complete, and the pericardium being a fibrous tissue does not stretch, and you are unable to get large effusion. Therefore, in cases of acute pericardial disease I should think the diagnosis would be difficult, because you would not be liable to have very much enlargement of the pericardium, as the effusion would be slight. I would like to ask whether in any of these cases there was a large amount of effusion? If so, I would expect such a case to be chronic.

I would attribute interference with the action of the heart to pressure upon the ventricles rather than to pressure upon the auricles. To my mind, the main muscle of the heart is the muscle of the ventricles. The auricles act largely as reservoirs, and might be compared to the gall-bladder. They take up the surplus blood, and if there is enough pressure in the pericardium to prevent the auricles from acting the blood would be pushed upward and be accommodated by the large veins outside of the pericardium.

Another question is that of temperature. I did not see all of these temperature charts, but what I did see did not appear to be hectic tempera-

tures, and it suggests a question as to the part which the pericarditis plays in death, and suggests the examination of other features of these cases. When the pleuræ are attacked we know they can have a large amount of pus and much disease, and yet the patients may live a long time. These cases of pericarditis have a very small surface, and the absence of hectic temperature points to the fact that pericardial sepsis may not have been the main cause of death, and for that reason it is interesting at all events to see what part the pericarditis had in the deaths.

Dr. D. J. M. Miller: Dr. Scott has mentioned cases of pericarditis in children. I am particularly interested in that subject, because this winter I saw a case of purulent pericarditis in a child four months old complicating enterocolitis. In conjunction with Dr. Gittings I reported the case to the Pediatric Society. In 364 autopsies at the Children's Hospital we found $2\frac{2}{10}$ths per cent. of purulent pericarditis, 1 per cent. less than those quoted by Dr. Scott as occurring among children in London. An interesting point is that of these 8 cases only 2 were recognized during life. The diagnosis, according to all authorities, is particularly difficult in children. The friction sound is mostly absent, and the most reliable sign of pericardial effusion, viz., enlargement of the cardiac dulness, is absent, because the effusion is small and very often is associated with left-sided empyema or pneumonia. In the majority of cases the diagnosis first comes as a surprise at the autopsy table.

I would like to say in regard to the site of puncture that Dr. Morse, of Boston, has recently reported a case of traumatic pericarditis in which he demonstrated that punctures could be readily made in the fifth right interspace.

Dr. Riesman spoke of the simulation of pneumonia by large pericardial effusions. Dr. Edsall has recently pointed out that when in large pericardial effusions there is bronchial breathing posteriorly and below the scapula the bronchial breathing disappears when the patient leans forward, while in pneumonia this would not occur.

Dr. Richard H. Harte: I am particulary interested in what Dr. Hare said in regard to the wounding of the heart and in reference to the diagnosis of pericardial effusion. I recall a case in which a man was stabbed with a large butcher knife, and lived for three hours; and I feel that if he had obtained competent surgical treatment it would have been a favorable case for suture of the heart.

Again, with regard to the diagnosis of pericardial effusion, I had the privilege of opening a pericardium in which we feared an old effusion, and we found a large dilated heart. The mere opening of the pericardium, just as Dr. Hare has said, did no harm, and the patient seemed better for the surgical manipulation. In one of the cases which Dr. Scott reported this evening, which was under my care, the question of diagnosis was difficult even when the pericardium was opened. It was only after two weeks that

I was able to verify whether the opening had been made in the pericardium or pleural sac by inserting my finger after removing a large drainage-tube. I felt at the time of operation that the opening was in the pericardial sac, which under these conditions is often very much modified.

Dr. J. C. Wilson: I would like to say a word or two in regard to the question of diagnosis. I believe that in certain cases the diagnosis between dilated heart and pericardial effusion is one of extreme difficulty. I should like to add to this another statement which I think is in accordance with the experience of most clinicians, namely, that rheumatic pericardial effusions of the usual serofibrinous character often attain a very large volume, so that the symptoms are very urgent, and yet undergo resorption without operative interference. Therefore, in the rheumatic cases it is justifiable to postpone operative interference for a considerable time. What I want to emphasize more than anything else, however, is this, that in view of the difficulty of diagnosis in certain cases, and in view of the innocuousness of carefully conducted surgical operations, an incision layer by layer, down to and into the pericardium, is justifiable. The more I observe these cases the more convinced I am that paracentesis should be laid aside as inadequate if not hazardous. In well-organized hospitals, or under circumstances where operation can be satisfactorily performed, I believe it is better in any case of suspected pericardial effusion, or in any case in which a reasonable provisional diagnosis of pericardial effusion has been made, and the symptoms are urgent, to subject the patient to operation for the opening of the pericardium rather than to perform exploratory paracentesis. If there be fluid of any kind, the pericardium has to be drained, and the operation would therefore ultimately become necessary for this purpose. If an error in diagnosis has arisen the patient is not much worse for the operation, and in cases in which pericardial effusion is not found to be present, a satisfactory recovery from this operation may be expected.

Dr. John B. Roberts: Before the discussion closes it might be well to state that Dr. Hare's conclusions from accidental puncture of the heart, which, he said, relieved the engorgement of a dilated heart, are in entire accord with the results about twenty years ago of Dr. B. F. Westbrook, who recommended that procedure in dilated heart. We know that phlebotomy is valuable in such conditions, when the right heart is overdistended. Dr. Westbrook advanced the proposition that it was better to draw the blood from the heart itself than from a vein. Dr. Hare has given us an excellent illustration of the value of that procedure. In 1881 it was proposed by me that in cases of doubtful diagnosis an incision be made, in order to prove that the symptoms were due to dilated heart rather than to pericardial effusion. It is delightful to hear Dr. J. C. Wilson, a physician, now advocate this line of procedure; but it is what might be expected from one who is so well known as the expert in internal medicine who first actively advocated surgical operation in intestinal perforation in typhoid fever.

Dr. Anders: The papers read as well as the discussion have been intensely interesting. I have been especially interested in the paper of Drs. Roberts and Riesman. They have set forth the diagnostic features of pericarditis ably and rather exhaustively. Personally, I do not think that the diagnosis of pericarditis is extremely difficult, provided that we practice palpation and auscultation in rheumatism and other conditions in which it is apt to arise as a complication, as we are usually able to detect the presence of the pericardial friction in this manner. In cases in which there is an absence of hypertrophy of marked degree we should also be able to diagnosticate the presence of an exudate by means of the physical signs. On the other hand, where pericarditis arises in the course of chronic cardiac affections we encounter serious clinical difficulty. In this connection I recall a case in point and seen recently, which occurred in the course of acute articular rheumatism in a young physician who had been suffering from marked aortic regurgitation and a massive left ventricle. A diagnosis of pericarditis was made on the detection of the pericardial friction. That disappeared with oncoming effusion, and then it was extremely difficult to demonstrate the presence of the exudate. The point I would emphasize is that, since if this condition is not recognized early it is extremely difficult of recognition on account of its latent nature in the stage of effusion; we should never neglect to palpate as well as auscultate the heart in any condition in which pericarditis is apt to arise.

Dr. W. L. Rodman: More than twenty years ago, while I had charge of a hospital in Kentucky, I aspirated what I supposed was the pericardium. The needle passed into the right ventricle, and a considerable amount of blood was withdrawn. I had exactly the same experience that Dr. Hare had. The patient, like his, was much benefited by the result. The cyanosis disappeared, and the patient made a smooth recovery. What we had diagnosed as a distended pericardium was an overdistended heart in connection with croupous pneumonia.

I think one of the essayists failed to mention another successful case of suture of the heart. I remember distinctly a report by Dr. L. L. Hill, of Montgomery, Alabama, about two years ago, in which he detailed a number of successful cases, one of his own.

Dr. Gibbon: I think Dr. Rodman is correct. Dr. Hill's case was the third success in America. Dr. Hill's case is included in Dr. Stewart's table. I overlooked it in giving to Dr. Stewart the third success.

BLOOD CHANGES PRODUCED BY ETHER ANÆSTHESIA IN BOTH MAN AND THE LOWER ANIMALS.[1]

By J. M. ANDERS, M.D., LL.D.,

AND

L. NAPOLEON BOSTON, A.M., M.D.

The effects produced upon the blood by the inhalation of ether as an anæsthetic are of special importance, since they have a direct bearing upon both surgery and general medicine. The results of the investigations into this subject have served in a measure, at least, as a guide to the selection of an appropriate anæsthetic—*i. e.*, whether ether, chloroform, or nitrous oxide should be used in given cases. The effects of the usual anæsthetics upon the kidneys, heart, and lungs have received much attention, but records appear to be few wherein the influence which they exert upon the circulating blood is presented. In our series of investigations we have selected perfectly healthy human subjects and persons upon whom slight operations were performed, and have also employed rabbits, noting the changes produced in the blood in all cases as a result of ether anæsthesia.

In a previous article one of us (Anders[2]) reported a series of experiments made upon two healthy female rabbits and a healthy human subject without the usual previous preparation for the anæsthetic.

The results of those earlier experiments showed clearly that "the fall in the hæmoglobin in primary anæsthesia reaches its lowest level

[1] Read June 1, 1904.

[2] Jaundice, with Reports of Interesting Illustrative Cases: a Contribution to the Toxic Forms of this Condition, American Journal of the Medical Sciences, April, 1903.

about twenty-four hours after the termination of the anæsthetic state; also that one-half of this fall takes place during the first hour after anæsthesia, the other one-half during the succeeding twenty-three hours. The blood-regeneration goes on quite rapidly upon the survival of the anæsthetic condition. The hæmoglobin, however, is restored more slowly than the erythrocytes; hence the cells are inadequately supplied for the time being with hæmoglobin. At the end of twenty-four hours the color index begins to rise rapidly, and it reaches the normal level after the lapse of approximately seventy-two hours in man. A few myelocytes, however, may still be present at this time."[1]

The human subjects employed were not purged before the administration of ether, and therefore any concentration of the blood (increase in the number of red cells) that was observed must have depended directly upon the anæsthetic. Again, the question of the interfering influence of associated disease is largely eliminated from our experiments; and this as well as the purging, appear to our view to be the common sources of fallacy in certain previous investigations; the latter condition (purging) increasing the number of erythrocytes, while the former (disease) was commonly associated with leukocytosis. It was previously pointed out by one of us (Anders) that the polycythæmia resulting from ether anæsthesia does not depend solely upon the preparatory treatment, including purgation, since this was purposely omitted in some of the cases belonging to the former series. The decidedly increased blood production, or hæmogenesis, immediately following the administration of an anæsthetic would appear to be a factor in bringing about the polycythæmia according to our previous experiments. In other words, as shown also by Da Costa and Kalteyer,[2] invariably etherization produces increased hæmolysis, following which there is rapid regeneration of cells, with an increase in the number of red corpuscles.

These observers, in their admirable monograph setting forth the results of a careful hæmatological study of fifty cases, recommend that the hæmoglobin should not register below 50 per cent. in cases in which prolonged anæsthesia or major operations are necessary.

[1] Loc. cit.

[2] American Medicine, March, 1901.

These authors also state that in one instance they administered ether when the percentage of hæmoglobin was only 24; but they strongly emphasize the fact that such procedure should not be ventured upon except by imperative necessity of a vital emergency. In three cases dying under collapse during anæsthesia, Mikuliez found only 15 per cent. of hæmoglobin present in the blood.

T. L. Chadbourne[1] in the study of twenty-one cases found that there was an increase in the number of leukocytes per cubic millimetre following anæsthesia. In his series of cases counts were made immediately after ether had been continued for a period of sixteen and two-thirds minutes. The average increase in the number of leukocytes was found to be 37.3 per cent. (the lowest 6 per cent., the highest 73 per cent.). Chadbourne made differential leukocyte counts in five of his cases, and found that this increase affected both the polymorphonuclear cells and the lymphocytes to an equal degree.

DIFFERENTIAL COUNTS.

No.	Count.		Increase per cent.	Time, minutes.	Differential leukocyte count.		No. of leukocytes counted.	
	Before.	After.			Before.	After.	Before.	After.
9	21,290	27,220	23	25	Polynuclear 83.8 % Mononuclear 14.0 % Eosinophiles 0.2 %	82.4 % 17.6 %	500	500
13	7,600	13,150	72	18	Polynuclear 71.1 % Mononuclear 28.4 % Eosinophiles 0.5 %	69.4 % 30.5 % 0.1 %	1000	1000
14	12,950	20,690	21	12	Polynuclear 69.8 % Mononuclear 23.2 % Eosinophiles 0.2 %	69.8 % 23.8 % 0.4 %	500	500
15	12,350	21,360	73	18	Polynuclear 82.8 % Mononuclear 16.0 % Eosinophiles 1.2 %	77.6 % 21.6 % 0.8 %	500	1000
18	14,000	17,000	21	15	Polynuclear 83.2 % Mononuclear 16.8 % Eosinophiles	85.8 % 16.8 % 0.4 %	1000	500

All of these patients were victims of disease, and the majority of them showed over 10,000 leukocytes per c.mm. before anæsthesia. It was also observed that the greater the preceding leukocytosis the

[1] Philadelphia Medical Journal, February 18, 1899, p. 390.

more marked the increase resulting from the administration of the anæsthetic. Operation, however, appeared to cause a slight reduction in the number of leukocytes in some instances at least.

Chadbourne in all his cases endeavored to eliminate the immediate result of operation, but it is to be remembered that he was dealing in great measure with pathological blood. From a review by us of the literature, all authors agree that pathological blood suffers far greater destruction as the result of ether anæsthesia than does normal blood.

A. von Lerber[1] has studied 101 cases, making the counts both before and after ether anæsthesia and found the leukocytes to be increased in 96 cases. The remaining 5 cases should not be included, since a high-grade leukocytosis existed before the anæsthesia was administered. In 35 cases the number of leukocytes was doubled and in 23 trebled, as compared with the number found before the administration of ether. In von Lerber's series, however, there was no definite time at which the counts were made either before or after the anæsthetic; neither was there any attempt to exclude the results which the operation possibly exercised upon the blood. In certain cases a blood examination was made twenty-four to thirty-eight hours before the administration of the ether.

In 1895, J. Chalmers Da Costa[2] made an extensive investigation of the action of ether upon the blood, and in concluding his valuable paper on the subject offers the following summary: "Etherization produces a marked diminution in the hæmoglobin in the blood. The red corpuscles and the hæmoglobin are especially affected in blood previously diseased. The white corpuscles show irregular changes which are not characteristic."

Hamilton Fish[3] has considered at length the blood changes that are known to follow the administration of ether with operation. Fish agrees that ether as an anæsthetic causes a reduction in the hæmoglobin and also changes in the red cells. He further advocates the view that an anæsthetic extracts oxygen from the oxyhæmoglobin and combines with the latter. It is claimed that oxygen is extracted from the corpuscles, and should the hæmoglobin register

[1] Medical News, March 2, 1895.
[2] Annals of Surgery, July, 1899.
[3] Inaugural Dissertation (Berne), 1896.

below 50 per cent. the corpuscles cannot afford the loss. It was found that in persons residing at an altitude of one mile the hæmoglobin was reduced from 12 to 15 per cent. during the first hour of anæsthesia.

Fish makes mention of the fact that leukocytosis is produced by anæsthesia, and that the increase in the number of leukocytes affects principally the polymorphonuclear elements. Mikulicz goes so far as to state that a general anæsthetic, not specifying either particularly, should not be administered where the hæmoglobin registers below 30 per cent.

Oliver[1] reports a series of observations made upon animals with a view to determining whether or not normal red corpuscles are changed by ether anæsthesia. From Oliver's experiments upon rabbits (each animal being kept under the influence of an anæsthetic [ether] for one hour) he found an average blood fall of 1.1 before anæsthesia and 0.98 after anæsthesia. It was further found that during anæsthesia the corpuscles appeared practically as normal. From the results of his experiments Oliver concludes that the administration of ether as an anæsthetic does not cause injurious effects upon the healthy blood corpuscles.

It is further admitted in Oliver's paper that ether may produce destructive changes in the previously diseased red blood cells. This observer advances the theory that examinations of the blood made before, during, and after the operations are not reliable, since certain hæmatological changes are the result of the operation.

The view that ether should not be administered as an anæsthetic in cases in which the percentage of hæmoglobin shows decided reduction is by no means a new one, but, on the contrary, reference to the subject is made in nearly every article furnished by the literature. The percentage of hæmoglobin regarded as necessary by the various authors in order to render ether anæsthesia safe ranges from 30 to 50 per cent.

ANIMAL EXPERIMENTS.

Experiment 1 (L. B. 37). Gray rabbit, female, apparently in perfect health, weight 2 kilos 130 grams, given ether twenty minutes. A small piece of gauze was used to form a cone which was saturated with ether, of

[1] Lancet, June 27, 1896.

which 6 drachms were employed. The animal's blood was counted ten minutes before ether was begun and at various intervals after the administration of the anæsthetic until the animal's blood had returned to the normal.

1. *Ten Minutes before Ether.* Red blood cells, 4,462,000; leukocytes, 16,000; hæmoglobin, 60 per cent.; no crenation; the leukocytes stained pale with hæmatoxylin.

2. *Twenty Minutes after Ether.* Red blood cells, 5,750,000; leukocytes, 24,000; hæmoglobin, 50 per cent. There were many basophilic cells; also normoblasts; poikilocytes, few, and a few highly stained microcytes.

Differential Count Twenty Minutes after Ether. Polynuclear, 44.5 per cent.; mononuclear, 2.0 per cent.; lymphocytes (large), 3.1 per cent.; lymphocytes (small), 38.3 per cent.; transitional, 0.8 per cent.; eosinophilia, 9.8 per cent.; leukocytic shadows, 1.5.

3. *Forty Minutes after Ether was Begun.* The red cells were 3,567,000; leukocytes, 24,000; hæmoglobin, 52 per cent. Many red cells stained heavily and many others were distorted, stained faintly, and showed indistinct margins. There was an increase in the large lymphocytes and basophiles. Polymorphonuclear leukocytes and small lymphocytes showed a less marked increase.

4. *Twenty-three Hours after Ether.* Weight, 2 kilos 10 grams (loss 120 grams). Hæmoglobin, 30 per cent. (loss 22 per cent.); reds, 4,000,000; whites, 8000. Many red cells were found almost devoid of hæmoglobin, appearing as nearly colorless disks whose margins were slightly stained. These cells were distorted at times, and were always swollen and did not display their normal central concavity. Other of the red cells were shrunken to appear as deeply stained spheres (microcytes). The general picture of the blood was that of a surface studded with overstained microcytes, understained macrocytes, and many poikilocytes. The normal sized red cells all displayed a rather large pale central spot. Occasionally red cells showing punctate areas of basic degeneration were seen. Few nucleated red cells (normoblasts) were present. Leukocytes showed nothing abnormal, although the lymphocytes were somewhat increased.

5. *Forty-five Hours after Ether.* Red cells, 4,500,000; leukocytes, 6000; hæmoglobin, 46 per cent. Changes practically those noted twenty-three hours after ether.

6. *Seventy-two Hours after Ether.* Red blood cells, 4,980,000; leukocytes, 10,660; hæmoglobin, 45 per cent. Polymorphonuclear cells predominent. Red cells well formed, stained readily, but were pale.

7. *One Hundred and Four Hours after Ether.* Stained specimens appear as normal. Red cells, 4,333,000; leukocytes, 10,000; hæmoglobin, 51 per cent. It will be seen that the blood of this animal did not remain normal, though the animal gave no other evidence of being ill.

8. *One Hundred and Eighteen Hours after Ether.* Red cells, 3,500,000; leukocytes, 21,600; hæmoglobin, 40 per cent. All the red cells were pale,

and many of them showed a special affinity for both the acid and basic stain (polychromatophilia). Other evidences of cell degeneration (as distortion and crenation) were also present. The leukocytes did not return to normal after anæsthesia, yet their relative proportions were maintained (as shown by the differential count of 600 leukocytes).

EXPERIMENT 2 (L. B. 41). On April 15th gave black female rabbit ether; weight, 3 pounds 3 ounces; 15 c.c. of the anæsthetic were given.

1. *Before Ether.* Red blood cells, 3,968,700; leukocytes, 4200; hæmoglobin, 65 per cent.

2. *Twenty-five Minutes after Ether.* There was no appreciable loss in weight. The red blood cells, 4,375,000; leukocytes, 6500; hæmoglobin, 55 per cent. There was present decided poikilocytosis. Microcytes and macrocytes numerous. Many of the red cells appeared as pale swollen disks which were poor in hæmoglobin. Other of the red cells stained unevenly, but true punctate basic degeneration was not present.

3. *One Hour after Ether.* Weight, 3 pounds. The red blood cells numbered 3,280,000; leukocytes, 8500; hæmoglobin, 48 per cent. Stained blood, as found at twenty-minutes after ether, except that the polynuclear leukocytes displayed many coarse brownish granules. Nucleated red cells were common.

4. *Twenty-seven Hours after Ether.* The red cells numbered 4,690,000; leukocytes, 6200; hæmoglobin, 43 per cent. All red cells pale; nucleated red cells (normoblasts) common.

5. *Fifty-eight Hours after Ether.* The red cells numbered 4,810,000; leukocytes, 6800; hæmoglobin, 64 per cent. The stained blood was found to be practically normal.

Second Anæsthesia of L. B. 41 : *May* 4, 11 *A.M.*

6. Seventeen days after the blood had returned to normal the red blood cells numbered 3,010,600; leukocytes, 8000, and hæmoglobin, 50 per cent.

The reduction of 14 per cent. in the hæmoglobin content, the moderate lowering of the number of red cells, and slight increase in the leukocytes, and gradual loss in weight, are features to be observed in nearly all rabbits after they have been detained in the laboratory for even a short period of time. The impoverishment of the hæmic tissue is probably the result of immediate surroundings, captivity, etc., and must needs be taken into consideration when attempting to draw definite deductions from animal experimentation.

7. *After Being under Ether for One Hour* (*May 5th*). The animal was in good condition; one ounce of ether had been given. The red blood cells numbered 5,120,000; leukocytes, 9200; hæmoglobin, 49 per cent. At first glance the effects of ether upon the blood appear to be confusing in this experiment and demand that we call special attention to one of the common sources of error in hæmatological research. The red cells have

increased from 3,016,000 per c.mm. to 5,120,000 (an increase of 2,104,000). This increase in the red cells is directly the result of cyanosis, and when we consider the number of red cells in each cubic millimetre of undiluted blood, and the percentage of hæmoglobin corelatively, it is apparent that each individual red blood cell is now carrying but little more than one-half the hæmoglobin it did before etherization. The white cells are also reduced in proportion to the number of red cells now present.

8. *After Being under Ether* 100 *Minutes.* One and one-quarter ounces of ether had been given (now discontinued). The red cells numbered 4,440,000; leukocytes, 13,600; hæmoglobin, 43 per cent. Leukocytes appear to be about normal; red blood cells were much distorted; showed considerable variation in staining properties; many staining pale, while others were overstained and deformed; but there was less variation in the size of the cells than was displayed twenty minutes after the administration of the ether had been commenced.

9. *Twenty-four Hours after Ether Anæsthesia.* The red blood cells numbered 3,820,000; leukocytes, 8000; hæmoglobin, 53 per cent. Cells fairly regular in size; poikilocytes rather common, and many crenated cells; leukocytes appeared to be normal.

10. *Forty-eight Hours after Ether* (*May* 6*th*). The red blood cells numbered 4,330,000; leukocytes, 6000; hæmoglobin, 50 per cent. (normal). Size of red cells uniform; crenated cells were uncommon; poikilocytes none. There were a few pale red blood cells.

11. *Nine Days after Ether* (*May* 13*th*). The red blood cells numbered 4,490,000; leukocytes, 6000; hæmoglobin, 50 per cent.

Third Etherization (*May* 16*th*).

12. *Before Ether.* The red cells numbered 3,920,000; leukocytes, 6200; hæmoglobin, 51 per cent.

13. *Under Ether Twenty Minutes.* The red cells numbered 4,470,000; leukocytes, 6000; hæmoglobin, 47 per cent. Here the rise in the number of red cells was again enhanced by cyanosis, which, in reality, indicates that the hæmoglobin for each individual red cell is greatly reduced.

14. *Under Ether One Hour.* The red blood cells numbered 4,200,000; leukocytes, 5600; hæmoglobin, 45 per cent. The red cells when stained displayed practically the changes noted at the second etherization, except that all changes were less decided, while the blood returned to the normal in about thirty-six hours.

EXPERIMENT 3 (L. B. 21), May 5, 1902. A yellow rabbit, male; weight, 4 pounds 5 ounces.

1. *Before Ether.* The red blood cells numbered 6,230,000; leukocytes, 13,000; hæmoglobin, 82 per cent. Films when fixed and stained with eosin and hæmatoxylin showed the red cells to be well formed and normally stained; leukocytes also appeared as normal.

2. *Animal under Ether Twenty Minutes.* (One-half ounce of drug used.) The red blood cells numbered 5,760,000; leukocytes, 6200; hæmoglobin, 76 per cent. The stained blood was normal except for a few feebly stained red cells, some of which appeared as mere shadows. This experiment differs radically from those previously related, since both the red cells and the leukocytes were reduced as the result of ether anæsthesia. The fact that the leukocytes numbered 13,000 c.mm. before the experiment may deserve special mention in this connection.

3. *Animal under Ether Sixty Minutes.* (One ounce of drug used.) The red cells numbered 5,430,000; leukocytes, 6600; hæmoglobin, 72 per cent. (loss 10 per cent.). Loss in weight, 1½ ounces. Stained blood showed decided poikilocytosis; cells stained fairly well, but displayed numerous unstained areas (punctate basophilia); microcytes and macrocytes were numerous. Pale cells previously noted present.

4. *Twenty-four Hours after Ether.* Animal's weight normal, and he appeared to be in perfect health. The red cells now numbered 5,210,000; leukocytes, 5250; hæmoglobin, 71 per cent. (loss 11 per cent.). In the stained blood, large lymphocytes and eosinophilic myelocytes(?) were rather common. The red cells were pale, and a few normoblasts seen.

5. *Forty-eight Hours after Ether.* The red cells numbered 6,310,000; leukocytes, 8660; hæmoglobin, 85 per cent. (2 per cent. above normal). The stained blood was as normal. In striking contrast to the previous results of ether anæsthesia upon the number of leukocytes per c.mm., this experiment does not show an increase, but a decrease, in the number of leukocytes. A similar result was observed in the case of one of our observations upon the human subject. (See Experiment 2.)

Second Experiment (May 16th, Eleven Days after First Anæsthesia).

6. The red blood cells numbered 4,730,000; leukocytes, 8000; hæmoglobin, 79 per cent. Stained blood normal. The animal was given ether twenty minutes (½ ounce being employed), when death ensued. Just prior to death the red cells numbered 7,800,000; leukocytes, 8000; hæmoglobin, 57 per cent. (a loss of 22 per cent.). This decided reduction in the hæmoglobin, coupled with an increase of 3,150,000 red cells per c.mm., was the only unfavorable sign and appreciable precursor of a fatal issue. This animal did not develop leukocytosis at either anæsthesia, and the proportionate relation of the different leukocytes was normal.

HUMAN EXPERIMENTS.

CASE I. (L. B., p. 42).—J. H., male, aged thirty-three years, apparently in in good health; was given ether without previous purging.

1. *Before Ether.* The red blood cells were 4,080,000; leukocytes, 7600 and hæmoglobin, 86 to 95 per cent., several estimates being made with different instruments. Stained blood was in every way normal.

2. *Under Ether Twenty-five Minutes.* The red blood cells were 6,150,000; leukocytes, 16,000; hæmoglobin, 79 per cent. No poikilocytes; microcytes numerous; no macrocytes found. All cells stained palely but evenly throughout. White cells showed an increase in the polynuclear elements.

3. *One Hour after Ether.* The red blood cells numbered 8,220,000, an increase of 2,070,000 cells per c.mm.; hæmoglobin, 74 per cent. From the degree of cyanosis present it is fair to attribute a liberal portion of this increase to this condition. Hand in hand with an increase in the number of red cells there was a loss of 12 per cent. in the hæmoglobin. It is at once apparent that 74 per cent. of hæmoglobin is an extremely low amount for a blood of such condensation, and that each red cell is extremely poor in coloring pigment. Leukocytes, 9800; the leukocytes, which rose from 7600 to 16,000 during the first twenty-five minutes, have now dropped to 9800, despite the extreme degree of blood concentration. Studying these figures correlatively, one is forced to believe that a true leukopænia exists. Occasionally cells were seen which stained palely; no crenated cells. Variations in size of cell decided. Differential leukocyte count: polymorphonuclear cells, 80.97 per cent.; myelocytes, 4.27 per cent.; transitional, 11.00 per cent.; large mononuclear, 1.11 per cent.; small lymphocytes, 2.22 per cent.; eosinophiles, 0.43 per cent

4. *Twenty-four Hours after Ether.* There had been no vomit, and the patient had taken food and claimed to feel well. The red blood cells were 4,576,000; leukocytes, 13,600, and hæmoglobin, 67 per cent. (loss of 19 per cent.). In the stained specimens there were seen many macrocytes, many of which stained poorly, some only as shadows, and these were liable to be distorted (poikilocytosis). Leukocytes mostly large, mononuclear and polymorphonuclear forms.

5. *Thirty Hours after Ether.* The red cells were 4,600,000; leukocytes, 12,000, and hæmoglobin, 78 per cent. (an increase of 11 per cent. in six hours).

6. *Forty-eight Hours after Ether.* The red cells were 5,700,000; leukocytes, 10,600; hæmoglobin, 80 per cent. There was an appreciable increase in the polymorphonuclear elements and in small lymphocytes. Two myelocytes were found. Eosinophiles and myelocytes were also present. Some of the cells stained irregularly, but there were no pale cells as previously seen.

7. *Seventy-two Hours after Ether.* The red cells were 4,780,000; leukocytes, 7400; hæmoglobin, 85 to 86 per cent.

Second Etherization.

8. *Before Ether.* (Patient not purged.) The red cells were 5,005,000; leukocytes, 6700; hæmoglobin, 80 per cent.; the stained blood appeared perfect in all respects.

9. *Under Ether Twenty Minutes.* The red cells were 5,310,000; leukocytes, 8200; hæmoglobin, 74 per cent. Few megaloblasts were present, but there was practically no distortion of the red cells; microcytes few; all red cells stained perfectly; white cells about normal relation, except for slight apparent increase in the eosinophiles.

10. *Under Ether Forty Minutes.* The red cells were 5,250,000; leukocytes, 12,000; hæmoglobin, 77 per cent. Ether discontinued.

11. *One Hour after Starting Ether.* Perspiration free and slight vomiting. The red cells were 5,120,000; leukocytes, 15,000; the gradual rise continued, reaching its maximum number at this stage; hæmoglobin, 72 per cent.

12. *Six Hours after Ether.* The red cells were 4,430,000; leukocytes, 11,000; hæmoglobin, 63 per cent. (a loss of 17 per cent.). Vomited four or five times in the afternoon. It has appeared to us that this feature (continued reduction) of the blood after ether anæsthesia is of the utmost importance, especially where a second anæsthesia is required, and that it is possibly accountable for a fair proportion of deaths following a primary anæsthetic. In this patient the red cells were also reduced, and both the erythrocytes and the hæmoglobin remained at this low ebb for twenty-four hours. The fact that no operation was undertaken is also to be considered in this connection.

13. *Twenty-four Hours after Ether.* The red cells numbered 4,660,000; leukocytes, 5800; hæmoglobin, 63 per cent.

14. *Forty-eight Hours after Ether* The red cells numbered 3,820,000; leukocytes, 5700; hæmoglobin, 70 per cent.

15. *Seventy-two Hours after Ether.* Vomited twice, and there has been moderate sweating. The red cells numbered 5,747,000; leukocytes, 8250; hæmoglobin, 73 per cent.

16. (May 16th.) Vomited yesterday and this morning, frequent perspiration, anorexia, and constant drowsing. The red cells numbered 3,920,000; leukocytes, 6000; hæmoglobin, 76 per cent.

17. (May 26th.) Vomited yesterday and this morning. The red cells were 5,010,000; leukocytes, 5000; hæmoglobin, 72 per cent. The patient recovered.

CASE II.—Mrs. A., aged thirty-two years, married, healthy, except for dysmenorrhœa. Vaginal examination disclosed some thickening of the right ovary and its tube, with stenosis of the os uteri to the degree that it was impossible to introduce a small probe. The anæsthetic was administered for the purpose of dilating the cervix. The patient was not prepared for the anæsthetic, and had been permitted to take a light breakfast at 8.30 A.M., and the anæsthetic was begun at 2 P.M. Just prior to the administration of the ether an examination of her blood gave: red cells, 5,275,000; white cells, 11,400 per c.mm., and the hæmoglobin, 84 per cent. Fixed and stained specimens of the blood revealed nothing abnormal. A differential leukocyte count gave: polymorphonuclear cells, 53 per cent.; eosinophiles,

1 per cent.; mononuclears (large), 11 per cent.; lymphocytes (small), 22 per cent.; lymphocytes (large), 8 per cent.; transitionals, 5 per cent.

Ether Sixteen Minutes. Red cells, 5,950,000; leukocytes, 10,200 per c.mm.; hæmoglobin, 78 per cent. There was no evidence of cyanosis at this time, and the patient appeared to be taking the anæsthetic well. The stained blood showed great irregularity in both the form and size of the red cells; their surfaces staining unevenly, though this feature was not prominent.

Ether Twenty Minutes. The red cells were found to number 6,400,000; leukocytes, 6200 per c.mm.; hæmoglobin, 70 per cent. At this time there was present considerable evidence of cyanosis, and despite the decided increase in the number of erythrocytes per c.mm., the hæmoglobin continued to fall, having lost 14 per cent. The stained blood displayed a high grade of poikilocytes, and there was present decided irregularity in the tinctorial properties of these cells. Many of the microcytes were deeply stained, while a smaller number of macrocytes were found to be very feebly stained. In many of the red cells staining normally, for the most part, there were irregular areas which remained unstained by the eosin. Certain other of the red cells were greatly swollen and appeared as mere shadows, surrounded by a marginal band of well-stained protoplasm. A differential leukocyte count gave: polymorphonuclear cells, 81 per cent.; large lymphocytes, 10 per cent.; small lymphocytes, 6 per cent.; transitionals, 3 per cent.

CASE III.—A female, aged fifty years, studied through the courtesy of Dr. W. Easterly Ashton. The woman had displayed some symptoms of endometritis for an indefinite period, but at no time had she exhibited evidence of anæmia. She had neither been purged nor prepared in any way for the anæsthetic, except that she had not received food in the morning. The blood was examined at 10.30 A.M., and the anæsthetic promptly begun. Red cells, 4,230,000; leukocytes per c.mm., 6800; hæmoglobin in 84 per cent. Stained specimens of the blood appeared to be normal. A differential count of 600 leukocytes gave: polymorphonuclear cells, 85 per cent.; transitionals, 10 per cent.; large lymphocytes, 4 per cent.; myelocytes, 0.5 per cent.; large mononuclear, 0.5 per cent.

After Ether for Sixteen Minutes. Red cells, 5,000,000; white cells, 14,000; hæmoglobin, 68 per cent. This fall continued, and at the end of twenty minutes registered 65 per cent. (a loss of 19 per cent.). The patient did not display appreciable cyanosis at the time of the taking of this count. Until the present there had been no operative interference and she had taken the anæsthetic very well. The reduction of 19 per cent. in the hæmoglobin and the increase of 8400 leukocytes per c.mm. are therefore attributable to the administration of the anæsthetic. A differential leukocyte count of 600 cells gave: polymorphonuclear cells, 63.5 per cent.; small lymphocytes, 29 per cent.; large lymphocytes, 4 per cent.; large mononuclear, 2 per cent.; myelocytes, 1 per cent.; transitionals, 0.5 per cent.

The red cells when stained with eosin and hæmatoxylin were found to be deficient in hæmoglobin, most of the cells being understained. Many others were distorted and swollen, their centres staining but slightly if at all, while their margins presented a narrow, rather well-stained band. In fact, the central pale spot normally present in the red cell was slightly enlarged in cells appearing to be otherwise normal. During the study of three specimen smears of the blood three nucleated red cells were found (normoblasts). Further observation on this case was impossible.

Case IV.—Observation made at St. Agnes' Hospital, through the courtesy of Dr. Laplace. It was impossible to conduct an examination of the blood prior to the administration of an anæsthetic. The patient displayed no evidence of anæmia, the anæsthetic being administered for the purpose of dilating a urethral stricture.

Ether for Sixteen Minutes. Red cells, 4,850,000; white cells, 13,000; hæmoglobin, 70 per cent. Stained specimens of the blood were found to present the changes observed in Case III. Some of the red cells showed numerous streaks and punctate areas disseminated throughout their protoplasm, which did not stain with eosin, and from which areas the hæmoglobin had doubtless been extracted. These punctate areas were slightly stained by hæmatoxylin, but the picture presented by the individual cells was not that of true punctate basophilia. One nucleated red cell was seen (normoblast). A differential count of 600 leukocytes gave: polymorphonuclear cells, 53 per cent.; eosinophiles, 2 per cent.; small lymphocytes, 14 per cent.; large lymphocytes, 10 per cent.; large mononuclear leukocytes, 16 per cent.; transitionals, 4 per cent.; myelocytes, 1 per cent.

SUMMARY OF FINDINGS.

Our experiments on animals showed that the hæmoglobin was reduced in every instance as the result of ether anæsthesia; this reduction varied somewhat according to the length of time the anæsthetic was continued. It was further noted that approximately 10 per cent. of hæmoglobin was lost during the first twenty minutes after the anæsthetic state was attained.

The hæmoglobin reaches its lowest point in from twenty-four to thirty hours after ether anæsthesia. Rarely, the oligochromæmia is extremely great, but here again practically one-half the total reduction of hæmoglobin content of the red cells was detectable during the first twenty minutes of complete anæsthesia.

It was the rule for animals to display a less decided loss in hæmoglobin during second and third etherizations than at the first ether narcosis, and it was further found that this loss was likely to be

restored within a shorter period of time than was that resulting from the primary anæsthetic.

In the human subjects, the reduction in the hæmoglobin was no less decided than in the rabbits, one-half or more of the fall in the coloring pigment taking place during the first twenty minutes of the anæsthetic state. This oligochromæmia was notable even in cases showing an increase of red cells per cubic millimetre; in fact, the rule was to find the hæmoglobin and the red blood cells increased at the expiration of twenty minutes.

Polycythæmia. Polycythæmia was always found to be more or less directly the result of cyanosis, and its extent was proportional with the degree of cyanosis. In all our experiments the number of red blood cells per cubic millimetre was increased after ether after a period of twenty minutes, and when cyanosis was evidenced this rise was seen to remain above the normal number of cells per cubic millimetre, until the anæsthetic was withdrawn and the lividity of the patient disappeared. That there has been an actual loss in the number of red blood cells per cubic millimetre as a result of the anæsthetic appeared reasonably certain, yet it was not possible at all times to demonstrate this feature satisfactorily.

Changes in the red blood cells as the result of ether anæsthesia were always present after the anæsthetic state was attained. When cyanosis was present and the blood found to flow sluggishly from the site of puncture, the individal red cell appeared thick and compact and its edges thickened. The central biconcavity was often obliterated from such cells.

In blood obtained during and after the ether narcosis, degeneration of the red cells was commonly present. This consisted in a simple pallor of the cells in the milder forms; while in the more decided instances (great loss of hæmoglobin) there were to be seen many microcytes which stained deeply, and a number of macrocytes whose protoplasm stained feebly. At times these large cells appeared as mere shadows surrounded by a well-stained marginal band of varying depth.

Small streaks and irregular patches of pallor were seen disseminated over the surface of many of the red cells, and outlined areas from which the hæmoglobin had been extracted.

True punctate basophilia as seen in lead-workers, was not well marked. At times, the large distorted red cells (macrocytes) displayed an affinity for both the acid and basic stains (polychromatophilia).

Nucleated erythrocytes were occasionally found.

LEUKOCYTES. Leukocytosis was found to develop in three out of the five animal experiments, and to reach its highest point in from twenty minutes to one-half hour following the commencement of anæsthesia. In some instances the number of leukocytes did not show any marked increase, while in a third class these cells were greatly reduced per cubic millimetre as the result of the anæsthetic. Where a reduction in the number of leukocytes per cubic millimetre was found to follow ether narcosis, it was likely to proceed to a leukopænia, resulting in approximately one-half the number of leukocytes originally present for the animal. While ether generally excited leukocytosis, this condition was of short duration—the leukocytes (per cubic millimetre) returning to the normal in from twelve to thirty hours.

Differential leukocyte counts were never found to show any points either of interest or value. In a few instances myelocytes were detected.

In the experiments upon human subjects, it was found that an increase of from one-third to double the normal number of leukocytes followed ether anæsthesia in one class of cases, this change taking place during the first twenty minutes of the anæsthesia. In one case they were slightly reduced in number; in all cases the leukocytes soon returned to the normal number per cubic millimetre following a return to consciousness. A differential study of the leukocytes disclosed nothing of positive clinical importance. Myelocytes (in small numbers) appeared in the human blood after ether anæsthesia.

APHASIA AND THE CEREBRAL ZONE OF SPEECH.[1]

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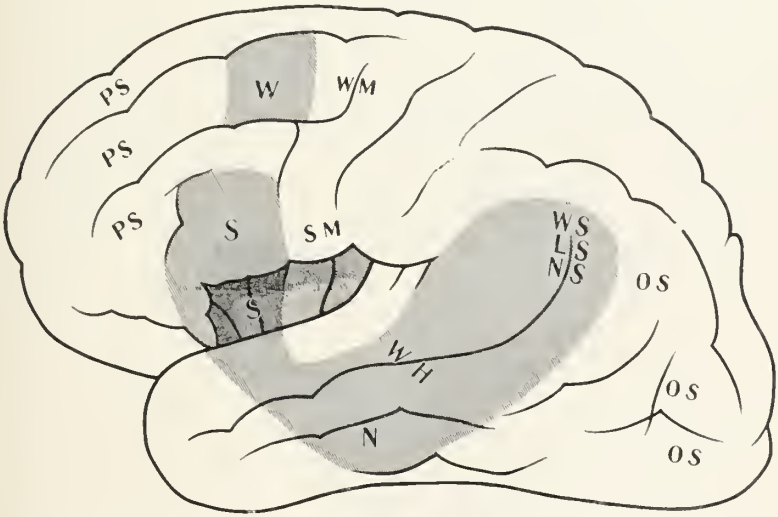
The cerebral zone of speech includes an extensive cortical region, probably not less than one-third of the cortical expanse of the lateral aspect of the left hemicerebrum, as shown in the shaded

Fig. 1.



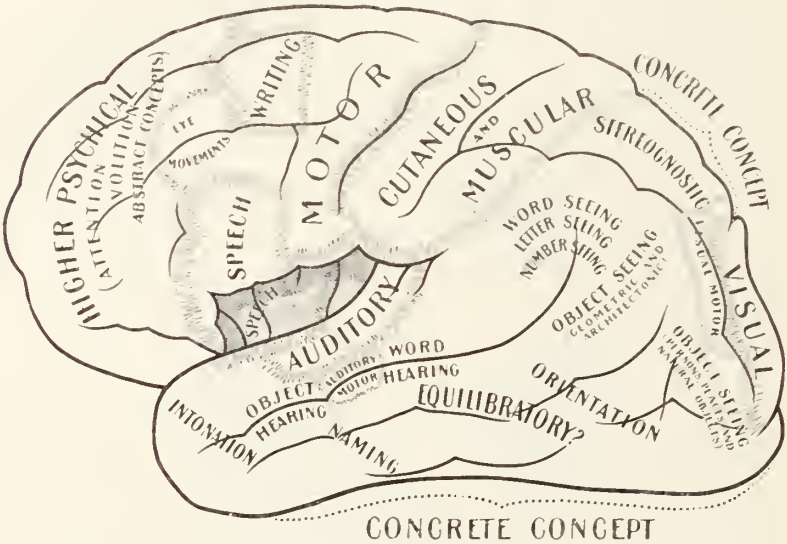
Cerebral zone of language: *W*, writing centre; *S*, *S*, speech centre (language arrangement for emission); *WH*, word-hearing or auditory centre; *WS*, word-seeing; *LS*, letter-seeing; *NS*, number-seeing; *N*, naming centre; *PS*, *PS*, *PS*, higher psychic region; *WM*, centre for movements of upper extremity concerned in writing; *SM*, centre for movements concerned in speaking; *OS*, *OS*, *OS*, area for object-seeing.

portion of Fig. 1. While for descriptive purposes it is best to designate it as a cortical area, it must be understood that both the cortex and subcortex of the entire region enter into its formation.

[1] Read June 1, 1904.

Within this great zone are several important centres or subareas concerned with particular attributes of language. In Fig. 1 these centres are *WH*, a centre for word-hearing, the auditory portion of the zone of language; at *S* and *S*, a great centre for speech, often called motor; at *WS*, *LS*, *NS*, a visual subarea concerned with the representation of words, letters, and numbers; at *W*, a graphic or writing centre, usually spoken of as the motor graphic centre; and at *N*, a naming centre in which it is believed by those who hold

Fig. 2.



Diagrammatic scheme of the physiological areas and centres of the lateral aspect of the human hemicerebrum.

to its existence that names are correlated with the concrete concepts of the objects named. In the diagram Fig. 2, in which the most important of the fundamental physiological areas of the cerebrum are represented graphically, these centres in the zone of language are more definitely isolated and designated by the terms word-hearing, speech, word-seeing, letter-seeing, number-seeing, writing, and naming.

Briefly stated, the auditive centre for speech is situated at the junction of the first and second temporal gyres about opposite the

point where the horizontal branch of the Sylvian fissure turns upward posteriorly; the dynamic or motor speech centre occupies the hinder portion of the subfrontal or third frontal convolution, and, according to the views of the writer, the insula or at least its anterior half; the visual centres immediately concerned with speech are located in the angular gyre; the site of the graphic or writing centre is in the caudal half or third of the second frontal convolution, and the naming centre is probably situated in the third temporal convolution.

Connecting these centres with each other, and also with other portions of the brain, are subcortical tracts composed of the processes of the cell bodies which are located in the cortex and of their fibrillary constituents. These associating tracts are of a complicated kind, although their number and arrangement are capable of analysis if sufficient time and study are given to the matter. Only one or two points regarding them need here be emphasized, namely, that the different centres are connected each to the other by a system of to-and-fro association, so that the impulses from special areas probably pass in both directions.

Surrounding the zone of language on all sides are centres and areas which have important relations to the function of speech. These are of at least three kinds, and an understanding of them may be reached by a study both of Fig. 1 and Fig. 2. On the sensory or receptive side of the brain are areas or centres concerned with such functions as object-seeing and object-hearing. Objects which are not recognized cannot of course be named, and these centres for object recognition, therefore, are necessarily contributors to the sensori-concept side of the zone of language. They are represented in Fig. 1 at the points marked as *OS*, and in Fig. 2 by the designation object-seeing, with subareas for geometric and architectonic forms, and for persons, places and natural objects. On the motor or emissive side of the cerebral speech zone are centres at *SM* and *WM* which are concerned with the execution of the work prepared for them respectively by the speech and writing centres. They are, in other words, the centres of representation of movements of articulation and enunciation and of the movements of the hand and upper extremity generally. At *PS* in the prefrontal portion of the

brain is a region where the highest psychical functions are especially represented. It is evident that in using language, attention, volition, and reasoning play a part, although this part may vary within wide limits. This higher psychical region, therefore, has its particular relation to the zone of language and especially to its dynamic side.

Broad, and it may be somewhat loose, comparisons may be helpful to the understanding of an intricate subject. The zone of language and that from which it is derived and with which it is correlated may be compared to the machinery and the methods by which one of our great natural products, like oil, is handled. The centres for object-recognition and the primary percept centres through which their memorial images are obtained may be compared to the producers or feeders of the system, the zone of language with all its centres and their connections to the refiners of the raw product, the executive motor centres and their projection fibres to the distributers, while the higher psychical region is the suzerain or director of the entire business.

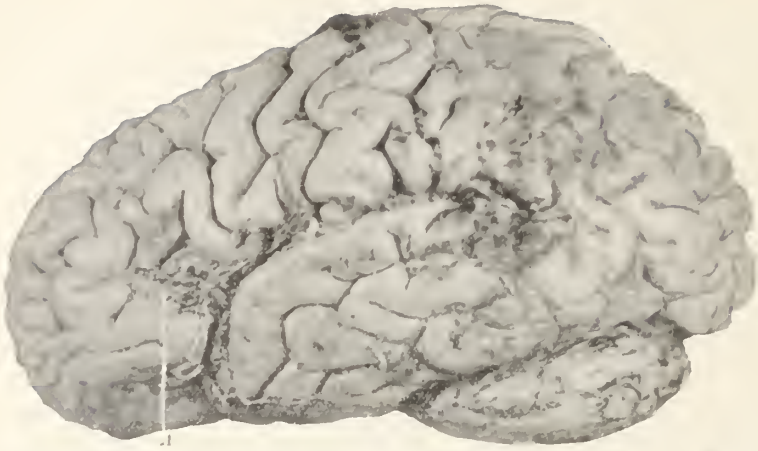
My objects in this paper are (1) to present succinctly a series of personal observations, some already recorded, and others not, demonstrating the existence of the centres of the zone of language according to the exposition just made, in this connection giving some corroborative proofs, although any extensive study of the enormous literature of the subject will be avoided; (2) to show by personal clinicopathological cases, with necropsies, the correlations of the zone of language with the sensory and motor spheres of the brain; and (3) leaving clinicopathological facts, to give briefly the biological principle which is fundamental to our conception of the speech zone and its subdivisions. That a discussion such as is intended in this contribution is called for at this time is evidenced in the first place because the extent and the particular subdivisions of the zone of language are still matters of dispute among those who have paid particular attention to cerebral speech. As high an authority in neuroanatomy and in clinical neurology as Dejerine, for example, still contends that a graphic centre in the second frontal gyre does not exist, holding that Broca's convolution is sufficient for all the purposes of the arrangement of language, both for articulatory

expression and for writing. The question of a naming centre may be regarded as still under discussion. Another matter of some interest and of some uncertainty is the exact part played by the subfrontal gyre and the insula respectively in the mechanism of language, whether, for instance, these parts represent stages in the evolution of cerebral language or whether they act conjointly as one centre. The writer has had the unusual opportunity of observing clinical cases with necropsies, and in some instances cases operated on by surgeons, which demonstrated every localization given in the zone of speech as here delineated and discussed. In presenting these cases I shall condense as much as possible, being desirous only of enforcing particular views without going into cumbersome details. References made to cases heretofore recorded will be especially brief. It will appear that however much I may have used the work of others in arriving at conclusions regarding the part played by the cerebrum in the faculty of speech, I have in every case been able to confirm the positions adopted or assumed by personal experience. In taking up the discussion of the different centres I shall proceed in part chronologically and in part in obedience to the natural development of the subject, first directing attention to the speech centre which has been longest known and is in most respects at least of greatest importance, that which is, in the writer's opinion, located conjointly in the subfrontal convolution and in the insula.

Two photographs (Figs. 3 and 4) are given of a case which has scarcely been exceeded in value in corroborating the existence of a centre or area of representation of speech in the caudal portion of the third frontal gyre and in the insula. Gordinier has referred to this case in one of his papers on agraphia, but without illustration, a photograph of the exterior of the left hemisphere and a brief history of the case having been furnished him by the writer.[1] At *A* in Fig. 3 is shown the external portion of an old hemorrhagic cyst, the only lesion related to the aphasia of the patient to be seen on the lateral aspect of the hemicerebrum. It will be observed that this area is strictly confined to the caudal portion of the subfrontal convolution. This brain has been sectioned only recently. A hori-

[1] Gordinier, H. C. Arguments in Favor of the Existence of a Separate Centre for Writing. American Journal of the Medical Sciences, September, 1903.

Fig. 3.



Lateral aspect of the left hemicerebrum, showing one portion of an old lesion circumscribed to the hinder part of the third convolution.

Fig. 4.

Horizontal transection of the brain, showing lesion of the insula continuous with that in the third frontal convolution indicated in Fig. 3; also atrophy of the entire left hemisphere, with enlargement of the ventricles.

zontal cut was made at the level of the upper surface of the insula, and revealed the lesion and the appearances shown in Fig. 4. It will be seen that the insula is largely destroyed. This proved on investigation to be a portion or a continuation of the lesion shown at *A* in Fig. 3. In other words, the third frontal gyre and the insula were neatly involved in the same circumscribed lesion, the second frontal convolution, the internal capsule, the basal ganglia, and all other portions of the brain not being the site of any old lesion. Although a section of the brain had not been made at the time of the publication of Dr. Gordinier's paper, his arguments in favor of a graphic centre based on a study of this case remain valid, as the second frontal convolution was not in the least involved.

I made many examinations of this case, and on a number of occasions used the patient at clinical lectures or demonstrations. The case was clearly one of aphasia with marked verbal amnesia and general disorganization of language on its dynamic side. Grammatical expression and combinations were lost. The patient could speak only in single words, or in short phrases, or very short sentences. What he said, however, he articulated and enunciated clearly; he had no paresis of the organs of articulation, enunciation, or vocalization.

Besides the old lesion causing the aphasia the necropsy showed several recent hemorrhages—a clot in the left parietal region and two subpial extravasations, one covering the right occipital lobe and another of small size along the longitudinal fissure on the left side, about three inches from the tip of the occipital lobe. A glance at the horizontal transection of the two hemispheres shows that the left hemicerebrum is much smaller than the right, the shrinkage appearing to be almost uniform for different regions of the brain. The ventricles and their horns are, however, not only relatively but absolutely larger than on the right side.

This patient, after an apoplectic seizure, developed aphasia, apparently without preceding paralysis or any other symptoms of focal lesion like visual blindness and hemianopsia. When examined by me about nine years after the onset of the aphasia he had almost complete inability to name persons and objects which he was able to recognize through all his special senses. He had also a marked form

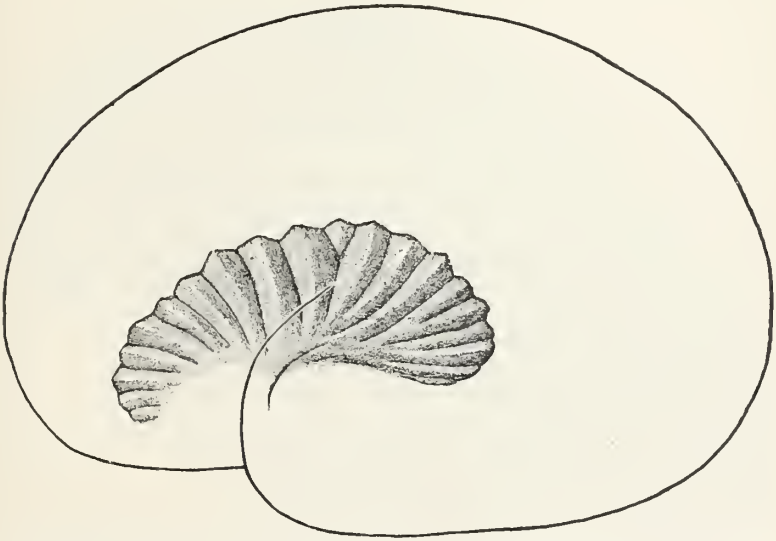
of paralexia. When he attempted to read, although he understood what he was reading, he repeated an absurd formula of a few phrases. He had limited spontaneous speech, even using short sentences without concrete nouns correctly. He could write many single words correctly, sometimes misspelling, however. He held his pen or pencil correctly and wrote with ease and firmness.

The part played by the insula in cerebral speech has long been a subject of discussion. In accordance with the plan of this paper I shall not go into the literature of the subject, but shall confine myself largely to an expression of personal opinion, giving the evidence for this opinion afforded by my own experience. Some of the most interesting data can be found in works on aphasia like those of Bateman and of Wyllie, and as regards recent literature, particular attention may be called to the paper by Dr. E. A. Spitzka on the "Redundancy of the Preinsula in the Brains of Distinguished Educated Men."[1] Some of the distinguished contributors to our knowledge of aphasia have even gone so far as to regard the insula as the sole cortical centre for speech. This view must, of course, be disregarded in the light of the numerous well-recorded cases of lesion of the subfrontal convolution with aphasia, and also when the numerous cases of degeneration or involution of Broca's convolution after lesion of the temporal lobe are considered. E. A. Spitzka holds that probably only the preinsula is concerned with speech, giving instructive personal observations and a review of the work of others in support of this position. His careful study of the brains of the Seguins, father and son, showed a redundancy of the left preinsula, also that the insulæ were marked by deeper fissures and a complementary plumpness of the gyres compared with ordinary brains. Exposure of the insula is usually regarded as evidence of disease or deficient cerebral organization, but Spitzka shows that this is not always the case, and gives what would appear to be the correct reason for the exceptions. While, on the one hand, deficient development of the opercula causes exposure of the insula, on the other hand in the highly intellectual, and especially in those with an unusual faculty of speech, a displacement and thrusting apart of

[1] Medical Record, June 15, 1901.

the opercula also results in insular exposure. Spitzka gives some facts both from comparative anatomy and from an investigation of human brains in support of the view that the caudal portion of the insula has a different function from its cephalic portion. He gives, for instance, two illustrations, one (Fig. 5) a sketch of the insula of the bottle-nosed dolphin, the other (Fig. 6) of a normal well-developed human insula. The differences are striking. In the case of the dolphin the fissures number about twenty, while in that of

Fig. 5.

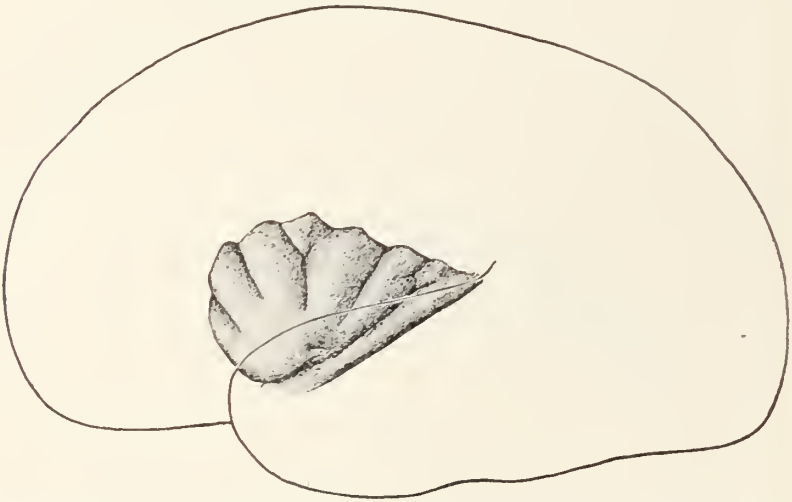


Insula of the bottle-nosed dolphin. The caudal portion of the insula of this cetacean is more redundant than the cephalic. The entire insula is large, having, as shown in the diagram, as many as eighteen fissures and nineteen gyres. (E. A. Spitzka.)

the man they number five main fissures, two of these being subdivided. The gyres of the preinsular portion of the dolphin's insula are relatively small, while the reverse is the case in the human insula. It seems to me that much is in favor of the view that the preinsula is particularly concerned with speech. Up to the present time the students of cerebral morphology, anatomy, and physiology have not succeeded in locating with any certainty the cerebral terminus of the vestibular portion of the eighth nerve, although the

writer has indicated it tentatively as probably being in the second temporal gyre. It may be that the postinsula is related to the cortical centre of representation of the vestibular nerve as is the preinsula to the auditory centre. Spitzka regards the preinsula as an association area for speech, a cortical region which unites the sensory side of the zone of language or a portion of it with the subfrontal gyre, which he speaks of as the emissary speech centre. The writer's view is somewhat different, if he understands correctly the position of Spitzka. I believe that Broca's convolution and the

FIG. 6.



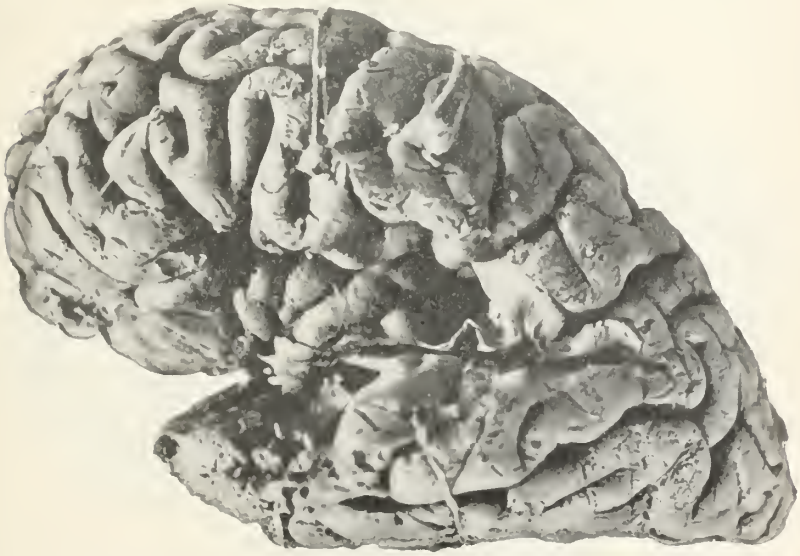
A normal well-developed human insula, showing that relatively the cephalic is more developed than the caudal portion of the insula. The number of fissures and gyres, as will be seen, is less than one-half as great as that in the dolphin. (E. A. Spitzka.)

insula (or perhaps only its anterior portion) constitute conjointly the same speech centre, a large cortical area being requisite for that portion of the mechanism of speech which is represented by this region. Neither of these areas has, I believe, projection fibres. Here language is organized for the use of the executive centres concerned with speech, in other words, for the facial, lingual, and laryngeal centres situated anterior to the foot of the central fissure. This view is similar to that of Clozel de Boyer.[1] The specimens

[1] Thèse de Paris, 1879, cited by E. A. Spitzka, Medical Record, June 15, 1901.

from the first case recorded in this paper (Figs. 3 and 4) support this contention, as when a circumscribed vascular lesion, either occlusion or hemorrhage, is limited to a certain area, it usually follows that the area affected has the same function. It is probable that the most important portion of the middle association area in the zone of language is situated in or near the midtemporal region, probably occupying portions of the second and third temporal gyres.

Fig. 7.



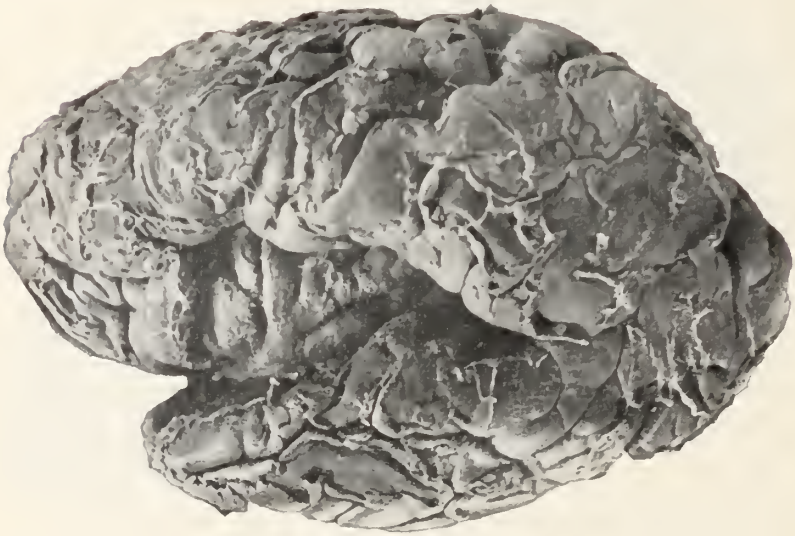
Photograph of the insula of a man of average intelligence without brain disease.

In one sense the insula and subfrontal gyre are undoubtedly association areas, but in them, to use the idea suggested by Broadbent, language is organized into propositions They constitute conjointly what might be termed a language-arrangement centre, but another association area concerned with the attachment of names to concrete concepts is probably situated in the temporal lobe.

In Fig. 7 is shown a photograph of the insula of a man of average intelligence, but not well educated, the opercula being pulled aside in order to focus the island. In Fig. 8 is given a view of the insula of an idiot.

In Fig. 9 is exhibited the insula of a well-known case recorded in 1891 by the writer, which helped to fix the position of the centre for word-hearing opposite the caudal extremity of the Sylvian fissure at the junction of the first and second temporal convolutions. The illustration shows extreme atrophy of the first temporal, retro-insular, and Broca's convolutions, with almost full exposure of the insula. In the light of what has already been said, these illustrations need but little comment. In the case of the man whose insula is shown in Fig. 7 no affection of language was present, the patient

Fig. 8.



Photograph showing the insula of a speechless idiot.

dying from spinal injury and its consequences; the idiot never acquired any power of speech, while the patient from whom the specimen represented in Fig. 9 was taken became word-deaf fifteen years before death, and nine years later totally deaf cerebrally because of a second destructive lesion of the right first temporal convolution and other parts.

The writer has long believed in the existence of a separate graphic or writing centre situated in the second frontal convolution, and has recently had the opportunity of obtaining new evidence of the

most positive character supporting this view. Two classes of cases may be regarded as proving the existence of such a centre, namely, (1) cases in which a lesion absolutely circumscribed to Broca's convolution, or the insula, or both, causes aphasia without agraphia, and (2) cases in which a lesion circumscribed to the alleged position of the graphic centre in the second frontal convolution causes agraphia without aphasia. I have had the opportunity of person-

FIG. 9.

Photograph of the insula of a case of lesion demonstrating the centre for word-hearing and the general auditory area. The original lesion was at *A*. The first temporal, retroinsular, and subfrontal gyres are much wasted. The first temporal convolution of the right hemisphere of this brain, with other parts, was also destroyed nine years before death by a second apoplectic attack, the first seizure causing the lesion at *A* having occurred fifteen years before death.

ally seeing two such cases. The case illustrated by Figs. 3 and 4 is, in fact, such a case, and I shall make no further reference to it, except to re-emphasize the statement that the patient was an aphasic but not an agraphic.

Recently in a case seen in consultation with Drs. J. W. McConnell, W. G. Spiller, and C. H. Frazier, the existence of a graphic centre or motor graphic centre, as it is usually described, was positively demonstrated. The case had many other interesting features. It

was one of tumor, largely prefrontal, removed successfully by Dr. Frazier at the Hospital of the University of Pennsylvania. I shall not go into details of the case, as I do not wish to forestall its fuller presentation by others, but shall, with their permission, call attention to the bearings of the case upon the question of the existence of a writing centre. The patient, a man twenty-nine years old, was first affected with a peculiar form of spasm attacking the face and head five years before coming under the observation of Dr. McConnell. Omitting all details of his history and the study of his case except in so far as it relates to his agraphia and his mental condition, it may be stated that for over two years he had been changing in disposition and failing in intelligence. A portion of a letter written by him in 1902 shows some omission of words and faults of spelling and construction, the same words in other parts of the letter being spelled correctly. These mistakes probably indicated the beginning of his agraphia. His recent letters were almost unintelligible. In consultation with Dr. McConnell I examined this man as regards his power to write, the day before the operation. All attempts to write, either spontaneously or from dictation, were absolutely futile, although he held and handled his pen and pencil properly and copied a sentence with comparative ease. Illustrations of his efforts to write his own name and other expressions will be given in the full account of his case. The patient was not an aphasic. He had considerable difficulty in articulating and enunciating, particularly for a short time before the operation, but numerous tests showed that this was due to paresis of the organs concerned with articulation and enunciation, and was in no sense an aphasia such as results from a lesion of the subfrontal gyre and insula, such, in other words, as was exhibited by the patient from whom the specimens shown in Figs. 3 and 4 were taken. The tumor in this case lay athwart the second frontal convolution exactly in the position indicated in Fig. 1 at *W* and in Fig. 2 at the position marked writing. The reader interested in the subject of motor agraphia is referred to the papers of Gordinier.[1] One case recorded by Gordinier is an almost perfect demonstration of the existence of the graphic centre.

[1] American Journal of the Medical Sciences, September, 1903.

Confusion still exists in the minds of many as to what is meant by the cerebral motor speech centre. Some, for instance, would make it include both the caudal portion of the third frontal gyre and the centres at the foot of the central or Rolandic fissure. As has already been indicated, the speech centre proper is circumscribed to the subfrontal convolution and insula. Here language is arranged for outward projection. Short association fibres connect this region with the true motor centres for articulation and enunciation, which constitute together the utterance centre or executive speech centre. Numerous physiological and clinicopathological facts and psychological principles could be readily adduced to uphold this view, but following the plan of the paper outlined, I shall content myself by referring to a single personal case and to one or two experimental observations. In 1889 I reported a case of paralysis strictly limited to the tongue, lips, and lower face, with slight implication of the palpebral orbicular muscle of the same side. Speech was entirely unaffected except as regards the effect on articulation caused by the lingual, oral, and slight facial paresis. The necropsy showed cortical softening involving the lower extremities of the central convolutions both on the lateral and Sylvian surfaces, and a spot one-half an inch in diameter about the middle of the internal portion of the insula. The softening reached into both the central and the precentral fissures. The anterior limit of the softening was one-fourth of an inch back of the anterior branch of the Sylvian fissure. This case seemed to clearly indicate that the true speech centre was confined to the subfrontal convolution and a portion of the insula, the area for the tongue, face, and allied movements being in the inferior portions of the central convolutions and largely in the precentral. In recent years I have come to believe that the motor region lies entirely or almost entirely in front of the central fissure. If there is any exception to this in the case of the human brain it is probably with regard to the centres for articulatory and allied movements. As is well known, the central fissure in the human cerebrum is only confluent with the Sylvian in extremely rare cases, and the entire gyral mass which separates its lower extremity from the Sylvian fossa may be concerned with movements which pertain to articulation, although by

far the largest portion of the motor region, even in this position, is probably cephalad of the central fissure.

Grünbaum and Sherrington[1] found that unilateral faradization of Broca's convolution in the anthropoids evoked no movement, and in one case observed by the writer, faradization of the human subfrontal during an operation gave similar negative results, although in the same case the application of the electrode to the centres of the face, eyelids, and head gave prompt and positive response.

It is not necessary to record here cases of lesion of the area for the upper extremity (*WM*, Fig. 1), or its subdivision, without involvement either of speech or writing. Such cases with necropsy have been seen by me, and a considerable number have been put on record. They simply show, as other cases referred to in this paper when discussing the writing centre, that the region of representation of the organization or arrangement of language for writing is independent of the area of representation of the movements of the upper extremity.

I shall make only brief reference to the auditory centre for speech, more commonly designated as the word-hearing centre. This is located at about the position *WH* in Fig. 1 and at that marked word-hearing in Fig. 2. As will be seen from a study of the diagram Fig. 2, the writer's present views with regard to auditory, visual, and concrete concept representation make it necessary to look upon some of the questions of speech localization, as of other forms of functional representation, somewhat differently from the manner in which these subjects have been hitherto regarded. In the cerebral cortex are situated primary auditory and primary visual centres, and, indeed, primary centres for all the forms of sensibility, special and more general, as for cutaneous sensibility, smell, and taste. Each of these centres has surrounding it what may be termed its sphere of influence, this in the case of the auditory centre being a large area concerned with all forms of what in a broad sense might be termed object-hearing. Within this general region of special sounds-recognition are probably located a separate centre for natural sounds, like the notes of birds, another for musical notes, and still

[1] Proceedings of the Royal Society of London, November 21, 1901; February 27, 1903; May 25, 1903.

another for word-hearing. The centre of primary recognition is probably located, as represented in the diagrams Figs. 1 and 2, in the caudal portion of the first temporal gyre and in the retroinsular gyres. The word-hearing centre is on the outskirts of this region, at about the junction of the first and second temporal convolutions. It is not necessary to reproduce the evidence of this localization, which has been accumulating for years, and especially since Wernicke's first thorough presentation of the subject in 1874.[1] No more convincing case indicating the localization of this centre has been presented than that reported by the writer[2] in 1891. This case is now well known, and has been referred to in various articles and treatises. The illustration Fig. 9 is from a photograph of the left hemisphere of this case. From a study of the lesions found in this brain I concluded that the centre or area for word-hearing was situated in the caudal thirds of the first and second temporal convolutions, the focus of the area being about in a line with the extremity of the Sylvian fissure.

Built up by processes of education and through heredity, visual centres for numbers, letters, and words have developed out of a general area for object-seeing in that portion of the brain usually designated as the angular gyre (at the positions indicated by *WS*, *LS* and *NS*, Fig. 1, and by the terms word-seeing, letter-seeing and number-seeing, Fig. 2). The evidence in favor of this highest visual area is of such a positive character and so well known that it is not necessary to adduce it in this paper. I would simply say that during the last two or three years I have had further corroboration of this localization in cases of brain lesion for which operations have been performed. In one of these cases the existence of a centre for object-recognition, independent of that for the recognition of words, letters, and numbers, was indicated by attacks of transient mind-blindness, the lesion in this case probably occupying portions of the second, third, and perhaps of the fourth occipital convolutions. At the point marked *N* in Fig. 1, and naming in Fig. 2, is probably located a special region where names are correlated to the concrete ideas of objects which are recognized by sight, hearing, or

[1] Der aphasische Symptomcomplex, Breslau, 1874.
[2] University Medical Magazine, November, 1891.

other senses, or through the interaction of the centres of these senses. While the separate existence of this centre may be regarded as not absolutely settled. I believe the weight of evidence and of *a priori* considerations are in favor of its existence. From the nature of the work which it is supposed to do its situation should be related more closely to the sensory than to the motor side of the brain, and a portion of the third temporal convolution, or of this and a portion of the second temporal, as indicated in the diagrams, would seem

FIG. 10.

Photograph of the lateral aspect of the left hemicerebrum of a case of hemiplegia and complete aphasia, the apoplectic attack having occurred between fourteen and fifteen years before death. All parts of the brain supplied by the left medicerebral artery are necrosed.

to be its probable location. Some clinicopathological facts also point to this conclusion, as, for instance, a case described by the writer and McConnell, and others recorded by German observers. The reader interested in this subject is referred to the writer's articles on aphasia in his work on *Diseases of the Brain*,[1] and to a paper published in 1902.[2]

The correlation of the cerebral zone of speech with the sensory

[1] The Nervous System and Its Diseases. Philadelphia, 1898.
[2] Journal of the American Medical Association, October 4, 1902.

and motor regions of the brain is illustrated by those cases of mixed sensory and motor aphasia which are not infrequently observed. A common symptom-complex is that of nearly complete right-sided hemiplegia with correspondingly almost complete aphasia. Two or three such patients presenting this syndrome are usually to be found in the wards for nervous diseases of the Philadelphia Hospital —the hemiplegics with a single recurring utterance or with only one or two such utterances. The photograph of a specimen recently obtained from such a case is shown in Fig. 10. The case is identical in most respects with one reported by Burr.[1]

My patient was for many years in the Philadelphia Hospital, and was frequently used by me and others to illustrate lectures and demonstrations of aphasia. She was one of a group of cases studied by me with reference to pantomime as well as speech, and a clinical report of the case was made in Vol. II. of the *Philadelphia Hospital Reports*, published in 1893, the patient living eleven years after that time, but not changing in any essential particular until her last illness. She was about fifty-one years old at the time of her death. Fourteen or fifteen years before her death she had an apoplectic seizure, causing loss of power in arm, leg, and face, without aphasia ; a few months later she had a second seizure, causing profound hemiplegia with contractures and aphasia. Sensation, so far as could be determined, was unimpaired. The deep reflexes in both upper and lower extremities were exaggerated on the paralyzed side. It was impossible to learn whether or not the patient was word-blind in the absence of her previous history. She was largely but not completely word-deaf. She was not mind-blind. "Her speech and pantomime," citing from the *Philadelphia Hospital Reports*, "might be said to be summed up in her *la-la* and laugh and a much less frequent expression of anger or displeasure, which was usually accompanied by a movement of covering her mouth with her hand. The appearance of her face could not be said to be without expression, but, like her recurring utterance, it was an appearance nearly always the same. It might mean that she was pleased and it might signify assent or dissent ; it might mean anything at all. Sometimes the *la-la* and the laugh became louder and more emphatic, but

[1] American Journal of the Medical Sciences, February, 1904.

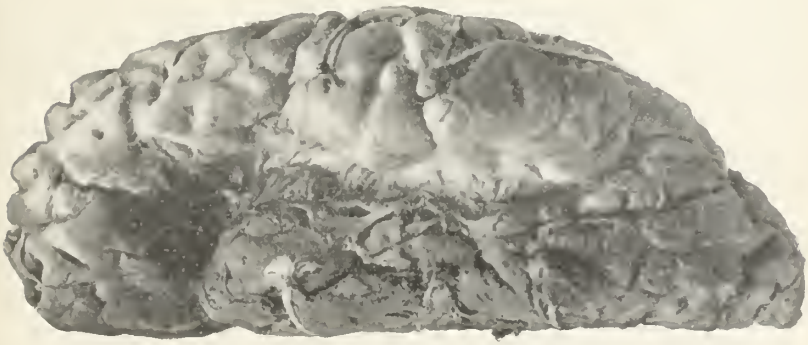
that was all. She never nodded the head in the usual manner for *yes* or *no.*" This patient died in 1904, during the term of service of my colleague, Dr. C. W. Burr, to whom I am indebted for the opportunity of obtaining the results of the necropsy, the record of which will be omitted except as regards the brain.

Examination showed that the left medicerebral artery was occluded near its origin from the internal carotid, and necrosis of all parts supplied by the branches of this vessel had occurred. The first temporal convolution, the insula, the subfrontal, precentral, postcentral and inferior parietal convolutions were all necrosed and atrophied, as were also the ganglia and capsules and corona radiata, in so far as these are supplied by the branches of the medicerebral artery distributed to the interior of the brain. The left hemisphere was greatly reduced in bulk and had the appearance of being caved in between the prefrontal and occipital regions. The external appearances are shown, although not particularly well, in the photograph Fig. 10. The whole hemisphere is greatly reduced in size, as shown both externally and in a horizontal section made at the level of the insula. The parts necrosed included the entire motor region and its environing sensori-concept-motor-speech zone, and in addition, large regions of the ganglia and capsules at the base. The lesions found in this case, as in that of Burr, are probably those usually present in cases of complete aphasia with a hemiplegia also nearly complete.

In some cases the aphasia is as nearly total as in the case just recorded, the hemiplegia being absent or represented only by an incomplete hemiparesis. In Fig. 11 is shown the photograph of the brain of such a case. This patient, like the one just recorded, was for many years a well-known character at the Philadelphia Hospital. Just as the other patient was frequently spoken of as *la-la,* so he came to be known as *anyone any,* this being his single recurring utterance. The history of the case is unfortunately lost, but the patient was well known to me, and was frequently used by me in clinical lectures and demonstrations. He was slightly paretic on the right side, but to so small a degree that he was quite useful as an assistant in one of the kitchens of the hospital, where he long worked. He was partially word-deaf. It is probable that he was completely word-

deaf at first, and had to some extent recovered word-hearing power. He was very acute in observing others when they were speaking, apparently sometimes recognizing what they said by the movements of articulation. To everything that was said to him he would simply answer *anyone any*, yet his appearance and manner sometimes indicated that he knew what was said, his knowledge evidently being obtained partly by sight and partly by hearing. His pantomine was not nearly as much disturbed as in the preceding case. Necropsy showed the conditions indicated in Fig. 11. The main trunk of the left medicerebral artery was not completely closed as in the preceding case, the branches occluded being those distributed to the

Fig. 11.



Photograph of the left hemicerebrum of a case of complete aphasia with right hemiparesis. The subfrontal convolution, the insula, and the first temporal convolution and adjoining parts are completely destroyed.

insula and to the opercular convolutions, the third frontal, central, subparietal, and first temporal; those convolutions were so necrosed as to be almost obliterated.

It has seemed to me worth while to record the cases contained in this paper and at the same time to glance at the entire subject of the cerebral zone of speech, with the view of clearing up some disputed points and emphasizing others. The acquirement of speech is the first great step in the separation of man from animals lower in the scale. Man differs from the gorilla chiefly in his ability to talk and in being better looking. Relatively the brain of the gorilla and of the other anthropoids shows lack of development in the region

given over in man to the zone of speech, while in these animals the primary centres for vision, audition, and movements are largely developed. The centres for word-hearing, word-seeing, for speech-associations and for writing have developed in the race and in the individual as these functions or attributes have been added to man's resources for communicating by language with his fellows. It is well that the students of comparative and of human anatomy and physiology, that clinicians and pathologists, should spend much time and labor in determining the existence and in accurately locating the various centres of representation in the zone of language, but the existence of these centres is ordained by evolution. The position, the size, and probably the shape of the cortical areas which are spoken of as the centres concerned with speech are determined by unalterable biological principles. The anthropoid and the human savage alike have large cerebral areas for object-recognition, whether through sight, hearing, touch, or the other senses. As man learns to talk the auditory speech area or centre develops in close proximity to the regions of primary representation of sound. Similarly, centres for visual speech develop within the sphere of influence of the primary visual centre, and the areas of language arrangement both for speech and writing in proximity to the great motor area, parts of which become the final cerebral instruments of speech. It is just as necessary that the man who writes spontaneously should have a centre for language arrangement for writing as that he should have a similar centre for speech, and the same is true of every subarea so far determined in the great zone of language. With our present knowledge of cerebral structure and function it is just as certain that a separate graphic centre or a separate centre for word-hearing or for word-seeing has its place in the human cortex on the principles here enunciated, as appears from the records of convincing clinicopathological cases by Broca, Gordinier, Eskridge, Dejerine, Wernicke, or anyone else.

DISCUSSION.

Dr. J. Hendrie Lloyd: I think that any person who stops to consider for a moment the subject of speech must realize that the organ of speech in the human brain is not a single organ. It is practically a fourfold organ. This can be appreciated when it is considered how we acquire language. The child at its mother's knee learns language through the auditory apparatus. It trains the auditory centre. It next attempts itself to make words, bringing into operation an entirely different series of neurons. That centre is located as shown on the diagram and is a motor centre. The next thing that occurs is when the child goes to school and is taught to read. All the ideas of language here flow through the eye, and that centre is apparently located in the neighborhood of the angular gyrus. The next attempt is to learn to write, which brings an entirely different nervous mechanism into play; that which is connected with the arm and which is supposedly located in the region marked, the upper part of the precentral convolution. We can better realize this fourfold mechanism when we consider that a man has a certain grasp of his mother tongue that he can never acquire with a foreign language. Professor Blakie, an eminent linguist and Greek scholar, was so exasperated time and again to find that his knowledge of the Greek language failed him at critical moments, that he studied this thing out in a crude way himself; shutting himself in a room every day, he declaimed Greek to himself, until his auditory centre was trained to appreciate and understand the Greek spoken language, and so he got a control he had not had before. We all know that while we may be able to read our French and German, we may not understand it when spoken, or be able to speak a word of it.

I am aware that the idea of a separate graphic centre is not accepted by some persons; that there is a disposition to deny the existence of this centre. This opinion by Dejerine I have always felt was too dogmatic. There is now one case at least on record (that reported by Dr. Mills) with lesion of the graphic centre, in which the patient was not able to write though he was able to speak. It is difficult to undertand how there could be no graphic centre if all the other methods of speech have each its own centre.

The most important part of Dr. Mills' paper, to me, is that part in which he speaks of object-seeing, object-hearing, etc., an association of the ideas of objects with these different mechanisms of speech. I can illustrate that subject best by saying that language is after all only a symbol of thought, words merely standing for things. We have a word for chair, for house. These are simply arbitrary sounds which have no meaning to anyone but ourselves; a foreigner associates no idea with them. These sym-

bols of thought are all accompanied by an association of ideas of the things themselves in our minds. This fact, as it is shown in aphasia, was brought out in one of my cases at the Philadelphia Hospital in a woman with a peculiar form of speech defect. Under certain circumstances she could not mention names. If I held an object before her eyes she could not name it; but if I were to put it into her hand, by a sort of reflex action she could name it. It seems evident that such a patient has aphasia of her visual centre; something has broken up the associations between her seeing centre, her object centre, and her speech centre. When I put the object into her hand, another avenue is opened. The impression gains admission through the avenue of common sensation, and thence to the motor centre for speech. That is probably at the point where Dr. Mills puts the area for cutaneous sensation. As soon as the sensation passes into her brain through that region, the association being complete between that region and her motor speech centre, out come the words. I think that feature is one of the most important and original portions of Dr. Mills' paper, and I think it deserves careful consideration in reference to this whole subject. The whole question is one of association between these various centres. My patient had a form of what is called mind-blindness. She could not recognize nor name an object when she saw it, but when she felt it she was able to give its name.

The difficulty of the subject is increased by the fact that in the human brain we are unable to do experimental work, and opinions are ever changing. Only a few years ago we were taught that the motor centres of the brain were on both sides of the fissure of Rolando. Sixteen years ago I reported at Washington a case of focal epilepsy in which I had demonstrated that the man had certain motor reactions when the current was applied to his post-Rolandic region. It was one of the earliest cases reported in America of such observations on the human cortex. Sherrington now tells us that that cannot be true, according to his experiments upon the higher apes. My observations were so positive that I prefer to hold that view until someone shows us the contrary from experiments upon the human brain.

There is one peculiarity about the aphasia which we sometimes see associated with Bright's disease. Neurologists are well aware that in uræmic states from Bright's disease, or contracted kidneys, we sometimes have curious focal symptoms of the brain, and among these is aphasia. I have seen several cases of uræmia in which aphasia was a symptom so marked that observers were almost ready to declare that there must be a focal lesion in the neighborhood of Broca's convolution. A few months ago I saw a gentleman, seventy years of age, with atheromatous arteries and contracted kidneys, who had had recurring attacks of aphasia. He was a professor in a theological seminary and still attempted to deliver his lectures. Two weeks before his death he passed into a condition of mild

uræmic toxæmia, and during this period his aphasia disappeared and he could talk with absolute fluency. In other words, the more he became confused in mind the more fluently could he talk. The explanation of this is difficult.

Dr. W. G. Spiller: I shall refer to a few cases which I think are interesting in this connection. In 1897 a patient was brought to the Polyclinic Hospital who had received a severe wound in the left side of the head, which was shown in the photograph taken at that time The symptoms were chiefly paragraphia and paraphasia. In the course of the eleven days following the examination he had regained a great deal of the function of speech. Hemorrhage in or near Brocha's area was probably the cause of the aphasia. The man returned a short time ago to the Polyclinic Hospital with convulsions confined to the right side of his face, but without aphasia. A cyst was found upon the brain by Dr. Stewart and was removed.

Last Thursday with Dr. Whiting I saw a most interesting case of aphasia in a lady of sixty or sixty-three years of age. She told me that she had had two other similar attacks, but her husband says she has had several. A sister also has had similar attacks. A week ago last Sunday this lady became suddenly unable to speak, and it was noticed shortly afterward that she was ataxic, and had lost the sense of position. If she tried to put a glass on the table she put it to one side, so that it fell to the floor. If she tried to pick up a pin she would put her hand to the side of the object desired. This lasted for a few days and then entirely disappeared. When I saw her on Thursday, four days after the attack had begun, she still had some loss of sense of position, but the aphasia had disappeared. I know only one other instance in which there was said to be hereditary aphasia. Several members of the family had aphasia. I think in the person I saw the aphasia was of vascular origin, but may have been uræmic.

The posterior part of the first left temporal convolution is believed to be the centre for word-hearing. I have seen at least two cases in which this was demonstrated; one, a case of Dr. Mills, in which the left first temporal convolution was shown at operation to be softened, and another a patient brought into the Polyclinic Hospital under the care of Dr. Steinbach, in whom the chief symptom was word-deafness. I saw the patient in consultation and recommended operation because of the word-deafness. Since then hallucinations of hearing have developed. Dr. Steinbach operated and found a large cyst involving the left first temporal convolution. These two cases and many others show that the posterior part of the first left temporal convolution is the centre for word-hearing. Yet I have removed recently a brain which is very puzzling. The case was under the care of Dr. Allyn and had been under observation for a long time. The patient could understand all that was said to him, and had very little difficulty in speaking. He could carry on a conversation well, but at times

he would be at a loss for words. He could write legibly, but rarely did write and occasionally was liable to omit words. His memory had suffered, but he read much. Dr. Allyn has shown me a letter written by this man, and there is no paragraphia and only a few mistakes. In his brain the first temporal convolution on the left side is completely destroyed, yet he heard all that was said to him and he did not have paragraphia. The second left temporal convolution may have had some function in word-hearing, and it is probable that the right first temporal convolution assumed the function of word-hearing. Byrom Bramwell has reported cases in which Broca's area was destroyed without causing motor aphasia.

Dr. J. D. McCarthy: In connection with the subject of temporary aphasia, to which Drs. Lloyd and Spiller have referred, I would like to call attention to the influence of arteriosclerosis in its production. I have been studying the subject of arteriosclerosis of the cortex and have gone over my series of specimens of some twelve or thirteen brains showing lesions of the brain centres in connection with endarteritis. My attention was called to the subject from a study of the case of a woman with advanced sclerosis of the brain, which, while very extensive, did not produce areas of softening. During the year before she died, she would have periods of paralysis of the right face and arm, which would last from two to six weeks, associated with temporary aphasia. A patient who died at the Philadelphia Hospital during the past winter, for two weeks previously developed paralysis of the face and arm with marked motor aphasia. I have several other cases in which the symptoms point to the same conditions, *i. e.*, arteriosclerosis exhaustion. In the two cases above referred to there was no evidence of a uræmic toxæmia. The patients died from the obliterated condition of the vessels of the brain and there were no symptoms of uræmia. I have concluded that many of these cases called uræmic hemiplegias and paralysis are not to be classed as toxæmias of the cortex, but that they represent malnutrition of the cortex, due to the obliterated condition of the vessels supplying the cortex. In the advanced cases, in which there is aphasia and paralysis, we have been confusing arteriosclerosis with toxæmic paralysis. The cases to which Drs. Lloyd and Spiller referred are probably of this type. The condition can be compared with the condition of the nerves in which there is marked sclerosis and intermittent exhaustion of the nerve (intermittent claudication). I think that the condition of the terminal paralysis which is persistent and not intermittent in cases of marked arteriosclerosis, with or without kidney disease, ought to be considered as due to arteriosclerosis with deficient nutrition of these cortical cells, and in that sense not of toxæmic origin.

Dr. Mills closes: Of course, this paper was not one on the relative positions and extensions of areas like the cutaneous and motor areas, nevertheless, that subject has considerable bearing upon the question of the zone of speech, and in this respect Dr. Lloyd's remarks are of interest. He

is inclined to antagonize the now nearly accepted view that the motor region is in front of the central fissure. If you faradize the third frontal convolution or the insular you will get no response in the way of motion unless with a very strong current. If you faradize the lower extremity of the precentral convolution you will get response. Under some circumstances you may get response from the postcentral gyre, although this convolution is not a region connected with motion. A tumor causing spasms puts the brain in its neighborhood in unstable equilibrium. In their experiments in front of the central fissure Grünbaum and Sherrington got motor response with a minimum current; behind the central fissure there was no response.

In reference to Dr. Lloyd's case, I would like to say that in those days we made one and one-quarter inch openings instead of the three and four inches we make to-day. The methods we adopted of outlining the position of the central fissure and of placing the operation were not exact, and it was a chance half the time, especially with a one or two inch opening, whether this was behind or in front of the central fissure.

I regard Dr. Spiller's case as one of great interest. His remarks in reference to Bramwell's case are also of interest. I expect to take up this subject at Atlantic City at the coming session of the American Medical Association in connection with a paper on the treatment of aphasia by training. The probabilities are that in the right half of the brain is a potential zone of speech, and it is constitutionally larger in some individuals than in others; it is more ready for use when the other gives out. This is the case in left-handed people. Not a few individuals are partly ambidexterous. One who is left-handed or one who is partly ambidexterous, who uses his right hand with more facility has a better opportunity for recovering his speech. My experience has been that cases with word-deafness, or word-deafness with word-blindness, recover from word-deafness at least partially sooner than cases with aphasia of the so-called Broca type recover from their speech defect.

ON HYPERNEPHROMA.*

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HISTORIES OF THE CASES AND SURGICAL TREATMENT, BY DR. KEEN.

My part in this joint paper will concern chiefly the surgical treatment of hypernephroma. Dr. G. E. Pfahler, who referred one of the cases to me, has kindly consented to consider the symptomatology and diagnosis, and Dr. A. G. Ellis, who has examined not only the specimens from the two cases herewith reported, but also another, a post-mortem specimen, to consider the pathology.

The term hypernephroma is so recent that it does not even appear in Morris' *Surgical Diseases of the Kidney and Ureter*, the preface of which is dated May, 1901. It was first employed, I believe, by Birch-Hirschfeld[1] in 1896.

The term is applied to tumors of the suprarenal gland and is derived from a Greek form of the Latin name of the suprarenal gland. The Greek form is adopted in order to correspond etymologically with the Greek termination "oma," indicating tumor. Küster uses also the name "epinephroma," a term of similar origin.

* Read November 2, 1904.

Such tumors arise from the adrenal tissue, whether in the normal position of the adrenal (*i. e.*, suprarenal gland) or as aberrant "rests." Prior to 1883 these tumors were called by many different names—lipoma, sarcoma, adenoma, angioma, angiosarcoma, adenocarcinoma, myxoma, endothelioma, etc. In the two cases here reported I did not recognize their real character by the naked eye even after they were removed, but believed them to be in the first case a sarcoma, an opinion in which Professor Weir concurred, and, in the second, a possible tuberculosis or sarcoma of the kidney.

Grawitz,[2] in 1883, was the first to recognize their real character as arising from the adrenal or hypernephric tissue, the title of his paper being "On the So-called Renal Lipomata."

It has been stated repeatedly that they are very rare. The statement should rather be that, up to the present time, they have rarely been recognized, and they are much more frequent than we have heretofore supposed. Bevan even says they are the most common form of malignant tumors of the kidney, and the same statement is attributed to Israel. It is not surprising that they should be frequent when we remember the wide distribution of supernumerary adrenals or "rests" of the adrenal tissue. These have been discovered in many parts of the body; in the solar and renal plexus; under the capsule, or in the substance of the kidney, or in the perinephric tissue; in the broad ligament; along the spermatic vessels; in the testicle and ovary; in the liver, the mesentery, and the inguinal canal. This is not to be wondered at when we remember that in the embryological development of the suprarenal gland it is in very close physical connection with the mesonephros or primitive kidney and the sexual organs. In fact, Holmes[3] states that "rests" of such tissue can be discovered in various parts of the genito-urinary tract in 90 per cent. of all post-mortems.

The surgical prognosis of such tumors varies greatly. Some of them never manifest any malignancy, while others are exceedingly malignant, so that, as Ramsay has pointed out, the average duration from the onset of the first symptom until death was from 6.7 months to 10.6 months, the shortest duration being six weeks and the longest three years. This is a much more malignant course than usual in other malignant growths. Happily in neither of the cases which I

report to-night is there any apparent malignant tendency. In Case I. nearly four years have passed since the operation, and the patient's health is excellent; in Case II. nearly eleven months have passed since operation and the patient is in better physical condition than ever before in her life.

The malignancy is shown either by local extensive invasion not only of the kidney, but of neighboring organs, or by metastasis. Kelly[15] and Bierring and Albert[4] have called attention to the fact that metastasis not uncommonly occurs by reason of the invasion of the renal vein by such tumors. In fact, in several cases there has been even thrombosis of the vena cava itself. This invasion of the venous circulation readily accounts for such a case as is reported by Clairmont,[5] in which the recurrence was in the bronchial lymphatic glands. In view of this possible mode of metastasis, the latter authors properly call attention to the importance of not handling the tumor roughly, either before or during the operation, and of tying the vein at as early a stage of the operation as is possible.

It is very doubtful whether in most cases a proper diagnosis can be made prior to operation, hence, as a rule, the operation must be an exploratory one, and only carried to completion in case the tumor can be removed in its entirety. If such extensive adhesions are found as to prevent entire removal, or if metastasis to other organs be discovered when the abdomen is opened, the operation should be abandoned. The operation does not differ from an ordinary nephrectomy either by the abdominal or the lumbar route, or a combination of both, as is so warmly commended by Morris. If the tumor is very large, it may be desirable even to resect the twelfth or the eleventh and twelfth ribs, though this will only very rarely be necessary.

Case I.—R. J. D., aged fifty-two years, of Bethlehem, Pa., was admitted to the Jefferson Medical College Hospital December 11, 1900. His father died at eighty-seven years, of an unknown cause; his mother of some intestinal trouble; one sister of tuberculosis. The only illness that he remembers is an attack of typhoid twenty years ago.

Three years ago he suddenly had an attack of hæmaturia at night. The next morning, while at his work, severe, cramp-like pains commenced and lasted for three days. They were so severe that they were only checked

by the free use of morphine. The pain radiated from the kidney to the head of the penis. He had no further attacks for a year, when a second one, less severe than the first, occurred. About this time he noticed a tumor below the border of the right ribs. This was freely movable, so that he could push it up under the ribs and down into the right iliac fossa. He has had a great deal of pain at irregular intervals. During the last two weeks the pain had greatly increased. He had passed blood at intervals ever since the disorder began. Within the last two weeks especially he has been troubled with almost constant and severe hæmaturia, and on one occasion had to be catheterized, when one and a half ounces of bloody urine with many clots were removed. The presence of the clots often produced trouble in voiding the urine. He usually had to rise two or three times during the night. Rest relieved almost all the symptoms; exercise increased them.

On examination I found a tumor extending from the median line to the flank and from the costal border to the crest of the ilium. It was only slightly movable. It measured 12 cm. vertically and 8 cm. horizontally. The right half of the abdomen measured 35.5 cm., the left 34 cm. On palpation, pressure was felt reciprocally, anteriorly, and posteriorly. Heart and lungs were normal. The urine showed a reddish, flocculent deposit, about 10 per cent., in the specimen examined, but without clots; specific gravity, 1019; acid; albumin $\frac{1}{10}$ moist layer; no sugar; urea, 1.8 per cent.; by the microscope a small amount of amorphous urates, a few squamous epithelial cells, and a large quantity of blood cells, but no tube casts or crystals; no tubercle bacilli were demonstrable by stain.

Operation in my clinic, December 19, 1900. Professor Robert F. Weir, of New York, had kindly consented to conduct my clinic on this day, and operated upon this patient with my assistance. So soon as the abdomen was opened both of us believed that we had to deal with a sarcoma of the kidney. The outer layer of the mesocolon was incised and the kidney delivered and removed; the pedicle was tied with a continuous suture. The wound was then packed with iodoform gauze and only partially closed. On the sixth day the stitch was removed loop by loop without hemorrhage. The patient was discharged February 18, 1901, in good health, with the exception of a very small sinus, which, undoubtedly, would heal spontaneously in a few days. The day after the operation his temperature rose to 101° and fluctuated from 100° to 101° until the fifth day, when it became normal.

October 15, 1904, I had a letter from him, saying that he is in excellent health, with no evidence of any recurrence after nearly four years.

Dr. Ellis reported as follows (Laboratory No. 1415):

Two tubes of glycerin-agar were inoculated with urine; in twenty-four hours an abundant growth was demonstrable, grayish-white in color and

extending over the whole surface of the medium. There was also abundant gas production noticed in the tubes. Spreads were made and stained with Löffler's methylene blue and by Gram's method.

Microscopically numerous bacilli were demonstrable, 1 micron to 3 microns in length and 0.3 micron in thickness, arranged and occurring for the most part in pairs and also singly. They decolorized by Gram's method. Together with this organism there is seen another bacillus, 1 micron to 2 microns in length, 0.6 micron in thickness occurring mostly singly, having rounded ends and more or less oval in shape and stained by Gram's method, corresponding to *urobacillus pasteuri*. Cocci are also seen which are identical both morphologically and tinctorially with staphylococci.

Diagnosis.—Bacillus coli communis; urobacillus pasteuri; staphylococcus pyogenes albus.

Dr. A. G. Ellis also made the following report upon the kidney:

Case I. (Laboratory No. 1416).—The specimen is a kidney strongly resembling the spleen in shape and 13 cm. by 10 cm. by 9 cm. in dimensions; the weight is 520 gm. The plane surface is covered by a rather firm, thick, smooth capsule, brownish-red in color, containing numerous dilated vessels. An oval depression, measuring 5 cm. by 8 cm. and corresponding to the renal pelvis, extends diagonally across the surface; the margins of this depression are rounded. Projecting from it is a mass of fat and also several nodules, each 2 cm. in diameter and dark blue in color. The convex surface of the specimen presents a marked lobulated condition, which includes bossed elevations 3 cm. to 5 cm. in diameter; distinct nodules, 1 cm. to 2 cm. in diameter and from 1 mm. to 5 mm. in height, some presenting a cauliflower appearance; and, finally, mere granular points may be distingnished. These varied projections are entirely subcapsular, the capsule in no way differing from that covering the plane surface. The color variations of the nodules are remarkable, varying from pale gray to deep blue or red, all shades between these being represented. In consistency some of the nodules are soft, as though cystic, while others are quite firm. Incision of the specimen meets considerable resistance, and exposes surfaces of varying composition. Medulla and cortex can be differentiated only at the larger end, where there is an area of renal structure 5 cm. by 2 cm. by 2 cm. In this is seen one pyramid, the overlying cortex varying from 3 mm. to 10 mm. in breadth. This area is separated from the remainder of the surface by a band of dense white fibrous tissue from 0.3 cm. to 1 cm. wide, and extending from end to end; from this, narrower bands of varying width project into the surrounding tissue in every direction, dividing the surface into irregular alveoli. These bands are especially adherent to the capsule of the specimen, and extend deeply into what is apparently the renal pelvis. The enclosed areas or

alveoli contain finely granular, softened masses, ranging in color from grayish-pink to red or blue. In a few, the contents are yellow and firm. The specimen was fixed in Heidenhain's solution, dehydrated, cleared and embedded in paraffin; sections were cut and stained by hæmatoxylin, with the addition of eosin or Van Gieson, toluidin blue, and Mallory's reticulum stain.

Microscopic examination shows the tumor to be divided into various sized, irregularly circular, or oval alveoli by broad bands of dense fibrous tissue, continuous with the capsule at certain points. In these bands are small areas resembling in miniature the alveoli enclosed by the stroma. The alveoli proper are occupied in part by a reticulum of fibrous tissue which transmits bloodvessels. Certain of these vessels are quite large and possess relatively thick walls, others are capillary in size and are bounded by endothelial cells only. The former, in many instances, appear to be dilated, though at such points they contain little or no blood. Lining the spaces formed by this reticulum are large cuboidal or polygonal cells. These are placed on the fibrous stroma, or in the case of capillaries, on the endothelium forming their walls, giving in the latter instances the appearance of double rows of columns. Each cell possesses a single large deeply-staining nucleus placed centrally or near the base of the cell. Nucleoli are usually, but not always, conspicuous. The cytoplasm in the greater number of cells stains faintly. It is represented largely by vacuoles that undoubtedly are spaces formerly occupied by fat. Some of the smaller reticular spaces are almost filled by these cells, the free ends closely approaching each other. Larger spaces contain detached cells and also red blood cells, in many of the alveoli blood being present in such quantities as to become the predominant feature.

Diagnosis.—Hypernephroma of right kidney.

Case II.—Mrs. W. H. P., aged fifty-four years, of Philadelphia, was first seen in consultation with Dr. G. E. Pfahler on December 12, 1903. Her father died of carcinoma of the face, and her mother of carcinoma of the breast. She has been more or less of an invalid for many years. Two years ago she suffered from marked indigestion, but made a fair recovery. Early in the winter of 1902 she suffered from two accidents, but it is not certain that they injured the kidney. In one she was thrown from a carriage; in the second she fell from a stool upon which she was standing. After this second accident she kept her bed for several days.

On December 12, 1902, after exertion connected with a social function, at which she ate some chicken salad, her present illness began with marked gastrointestinal symptoms, such as nausea and occasional vomiting, alternate diarrhœa and constipation, and vague abdominal pains, accompanied with languor, progressive weakness, and anæmia.

In January, 1903, a small polyp was removed from the cervix uteri by Dr. Elizabeth L. Peck. Dr. Peck at the same time discovered a movable

tumor in the right lower abdomen, which she thought was probably a floating kidney. Dr. Pfahler was first called to see her early in November, 1903, and immediately took careful skiagraphs of the abdominal tumor. In the skiagraph the shadows were first thought to be gallstones, but later Dr. Pfahler was disposed to think that the tumor was a kidney with a suspicion of three stones in it.

The blood examinations were as follows:

	Erythrocytes.	Leukocytes.	Hæmoglobin.
November 23 . . .	3,920,000	12,000	40 per cent.
November 30 . . .	4,200,000	10,000	41 "
December 14 . . .	4,690,000	8,000	42 "

The urine was negative. She had never had an attack of hæmaturia. There was some mucus in the stools.

A few days prior to my examination she had vomited some undigested food, in which Dr. Pfahler found no free HCl. Owing to her great weakness he had not administered a test-meal. In the region of the pylorus there seemed to be a painful spot, with possibly increased resistance, but this was rather vague. She has never had renal colic. Her pulse has usually been from 80 to 90; her temperature from 100° to 102° of late. Her normal weight was 113 pounds, but this had fallen by November to 97 pounds, and when I saw her December 12 it was probably below 90 pounds. Her weakness had progressively increased so that she was very feeble.

On examination I found a rather slender, delicate woman, evidently very ill, but who faces her grave condition with great equanimity, and has made up her mind that an operation is necessary and shall be done. As soon as the abdomen was exposed a tumor was perceptible to the sight on the right side of the abdomen, extending from the right iliac fossa to the border of the ribs, and from near the median line into the right loin. Reciprocal pressure was felt in front and behind. The tumor was not particularly painful, and was fairly movable. No tumor about the pylorus or gall-bladder could be made out, nor was there any great sensitiveness. My diagnosis was a kidney enlarged to nearly if not quite double its normal size by the presence of a tumor. I advised an abdominal section. The evidence for disease of the stomach was not convincing, but if there was any serious condition found, it would have to be operated upon.

Operation.—December 16, 1903, by an incision in the right semilunar line. As soon as the abdomen was opened, I drew out the stomach, which seemed to be entirely normal. In the kidney a large tumor was perceptible just above its middle. It was lobulated, and in my opinion probably a sarcoma. The kidney was delivered through an incision in the outer layer of the mesocolon. The pedicle had to be tied in several sections; one enormous renal vein, at the upper part, the size of a finger, gave me con-

siderable trouble, and required three ligatures before it was completely secured, the final one being very near to the vena cava. As a further precaution against later hemorrhage, I packed some iodoform gauze over the stump. The distal end of the ureter was tied, cauterized with pure carbolic acid, and dropped. The mesocolon was then closed, except where the packing found its exit, and the operation terminated.

The operation was done on a hot water mattress. During the operation, Dr. A. B. Craig infused a quart of saline solution with a dram of adrenalin, 1 : 1000. In spite of her great weakness, whether due to these precautions or not, there was practically no shock after the operation, the temperature only falling from 99.2° to 98°, and the pulse rising from 109 to 118.

Blood examination on December 30:

Erythrocytes	4,910,000
Leukocytes	8,800
Hæmoglobin	42 per cent.

On the third day the packing was removed. Her temperature never rose above 99.6°, saving at 4 p. m. of the day of the operation when it rose once only to 103.2°, and she made an uninterrupted recovery.

The following table shows the secretion of urine from the left kidney:

First twenty-four hours	9½ ounces.
Second twenty-four hours	15½ "
Third twenty-four hours	12½ "
Fourth twenty-four hours	13¼ "
Fifth twenty-four hours	16¼ "
Sixth twenty-four hours	12½ "
Seventh twenty-four hours	20½ "
Eighth twenty-four hours	14¼ "
Ninth twenty-four hours	18½ "

To all of these amounts should be added more or less urine passed with the stools. Two culture tubes inoculated from the kidney showed no growth.

I first thought that the tumor was a sarcoma. Later, on making a section of it, I was inclined to think it was possibly tuberculous. The histological examination by Dr. Ellis, however, as he will explain, showed it to be a hypernephroma.

By February 15, 1904, two months after the operation, she had gained 30 pounds in weight.

On January 26, 1904, the blood examination showed:

Erythrocytes	4,910,000
Leukocytes	8,600
Hæmoglobin	56 per cent.

By May 14, 1904, her hæmoglobin had risen to 70 per cent.

On October 20, 1904, I saw her in blooming health. Her weight had increased to 136 pounds as contrasted with less than 90 pounds ten months

before. Indeed, she asserts that she is in better health than she has ever been before. The urine varies from 36 ounces in hot weather to 60 ounces in cold weather. The average is about 46 ounces.

Dr. Ellis made the following report upon the kidney :

Case II. (Laboratory, No. 2546).—The specimen consists of a kidney 11 cm. long, 5 cm. wide, and 3.5 cm. in maximum thickness. The weight is 298 gm. The organ shows no conspicuous gross lesion, except on the posterior aspect of the upper half where there is a projecting tumor, irregularly globular in outline, measuring in its greatest diameter 7 cm.

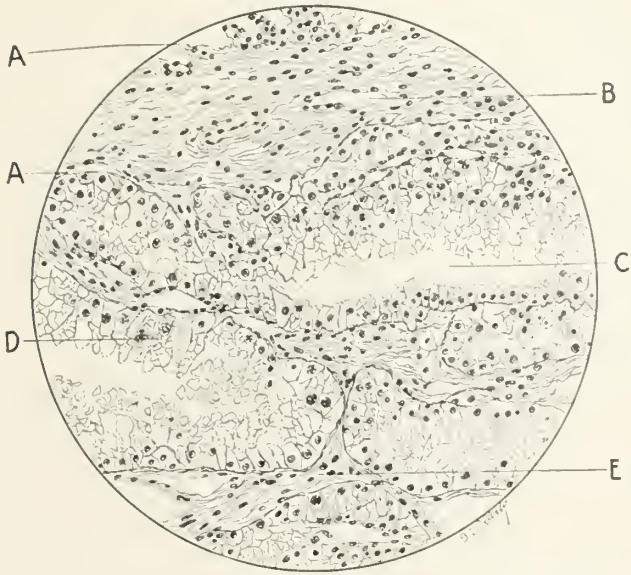
Fig. 1.

Hypernephroma, Case II. A, capsule of kidney extending over tumor; B, areas of hemorrhage.

It projects beyond the surface of the kidney a distance of 4 cm. The tumor is covered by a capsule, which is apparently an extension of the capsule of the kidney. It is very thin, translucent, and contains numerous small, distended bloodvessels. Broadly speaking, the surface of the tumor is fairly smooth, though it presents several rounded, elevated areas, varying from 0.5 cm. to 2 cm. in diameter. These areas have a maximum elevation of 0.6 cm. and merge gradually with the surrounding surface. They are much softer than the intervening tissue, which is quite firm, though readily compressible by the finger. The color of the tumor mass beneath the capsule is distinctly yellow. A few punctate dark areas resembling small extravasations of blood are scattered over the surface. Externally, the

tumor is sharply differentiated from the substance of the kidney and a mesial incision, which has been carried through the mass into the renal pelvis, shows the entire tumor to be thus clearly outlined (Fig. 1). Both cortex and medulla of the kidney have been usurped by the growth, which extends to the renal pelvis, without encroaching upon or notably projecting into that structure. The depth of the tumor as measured from the external surface to its termination at the pelvis of the kidney is 7 cm. The upper border of the growth is, at its junction with the surface of the kidney, 2 cm. from the extreme upper end of that organ, but its free extremity projects

FIG. 2.



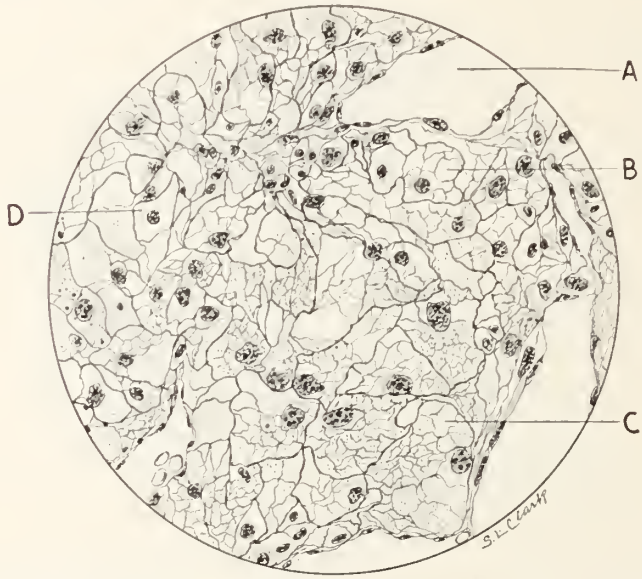
Section of hypernephroma, showing capsule (Case II). A to A, capsule; B, bloodvessel in capsule; C, alveolus surrounded by polyhedral cells, many of which are columnar and possess the general characters of the cells found in the adrenal cortex; D, typical spongiocyte; E, connective-tissue reticulum.

beyond the attachment until the outer portion is even with the margin of the kidney. The incised surfaces of the growth are yellow in color. They are marked out into variously sized circular or oval areas by narrow bands of grayish tissue, presumably fibrous in character. These areas exhibit pronounced bulging. Minute reddish points are seen dotting these surfaces, apparently marking the situation of several bloodvessels. Further incision of the mass reveals the presence on the surfaces of dark areas 0.5 cm. to 1 cm. in diameter. A few areas of softening are noted. The entire tumor mass, on its incised surface, is soft and very friable.

Slightly below the centre of the posterior surface of the kidney and a little internal to the common border, at a point 2 cm. from the margin of the tumor described, is a yellowish, slightly elevated nodule 0.5 cm. in diameter. It is immediately beneath the capsule of the kidney and is quite dense in texture. This nodule was removed entire, with the surrounding portion of the kidney, for further study, hence its depth of penetration was not at this time noted.

Inoculations were made and cover-glass spreads prepared from the tumor; part of the latter were fixed in absolute alcohol. Half of the

FIG. 3.



Section of hypernephroma, showing character of cells. Same section as Fig. 2 (Zeiss 2 mm. homo. im.). A, bloodvessel; B and C, spongiocytes. In the upper cell (B) the intraprotoplasmic spaces appear empty; the corresponding areas in the lower cell (C) contain faintly acidophilic fine granules. D, similar cells, with structureless clear protoplasm.

specimen was preserved by Kaiserling's method. The other half was fixed principally in Bensley's fluid, but small portions were also placed in absolute alcohol and in 10 per cent. formalin.

Examination of spreads stained by methylene blue shows them to contain a variety of cells. Polynuclear leukocytes are quite numerous as are also lymphoid and hyaline cells. In addition to these are large polyhedral cells ranging from 12 microns to 40 microns in diameter. The cytoplasm is finely or coarsely granular. The nucleus is large and usually single, though occasionally two nuclei are noted and in one cell four are present.

Some of the nuclei stain deeply, but many are but little darker than the cytoplasm. One or more nucleoli are usually present. A few of these cells, particularly the smaller ones, contain one or more vacuoles. In certain others a clear space separates the nucleus from the cytoplasm. Inoculations on agar yielded no growth.

The fixed portions of the tumor were embedded in paraffin and sectioned. Sections were stained by hæmatoxylin and eosin, hæmatoxylin and Van Gieson, toluidin blue, Mallory's reticulum stain, and by specific stains for glycogen.

Microscopic examination of the sections show the following characters: Sections including both the kidney substance and tumor show these two structures to be separated from each other by a distinct zone of fibrous tissue. This zone is quite wide and shades off gradually into the kidney substance proper, appearing to be a condensation of the renal tissue, with some added fibrosis, rather than a true capsule of the tumor. The structure of the tumor at this point is that of an exceedingly scanty, almost filamentous, fibrous stroma or reticulum, which ramifies in various directions as long bands, to each side of which are attached large cells, or form variously sized alveoli lined by similar cells (Fig. 2). The cells vary in size, but the majority are extremely large. They are cuboidal or columnar in type and contain round, fairly deeply staining nuclei, situated usually near the middle of the cell. The cytoplasm is very loose in structure and stains indifferently. It contains in many of the cells numerous clear or translucent areas that suggest the presence of fat, but which do not have the regular, clear-cut outline usually presented by distinct globules of that material. In many areas these cells are but one layer in depth, but other portions are made up of alveoli practically filled by several layers of them. In such areas the limiting membranes of the cells are the most conspicuous feature, forming an irregular network in which is seen the slightly granular, shadowy cytoplasm (Fig. 3). Many of these cells contain no demonstrable nucleus. Portions of the tumor are quite compact, while others possess an exceedingly scanty network of stroma and cells, the large spaces thus formed containing in some instances blood, in others detached tumor cells, both showing various degrees of cytolytic change. The parts of the tumor described in general structure and character of the cells resemble the zona glomerulosa of the adrenal gland, showing here, of course, cellular proliferation and also degenerative changes. In other sections are seen areas made up of columns of cells, forming a network, in the interstices of which are capillary bloodvessels. These cells are much smaller than those previously described, containing vesicular nuclei and clearly defined fat spaces. The similarity of these areas to the zona reticularis of the adrenal is very striking. Sections stained for glycogen by the iodine method show numerous various sized granules or droplets of that material in the large cells of the tumor.

The kidney shows granular changes in the epithelium of the tubules, accompanied, in some areas, by considerable desquamation. Slight recent hemorrhages into the tubules or tufts are occasionally noted, but are not at all conspicuous. A few granular tube casts are *in situ*. The capsule of the organ is slightly thickened and small areas of beginning fibrosis immediately beneath it are observed. The conspicuous fibrous transformation in the vicinity of the tumor has been previously mentioned; parenchymatous change is also more noticeable as the tumor is approached.

Sections through the small nodule in the kidney, mentioned in the gross description, show it to be beneath the thickened renal capsule. It is made up of a loosely arranged network of fibrous tissue enclosing spaces which, in the central part of the mass, contain cells resembling to some extent those in the large tumor. Dense accumulations of lymphoid cells are also present. Although it cannot be definitely stated that this is a metastasis from the larger tumor, it is reasonable to suppose that such is the true nature of the small nodule. It is not sharply demarked from the adjacent tissue, possesses the semblance of a capsule at points only, and shows, equally with the larger mass, such evidences of growth as to indicate that both are progressing at about the same rate; if any difference can be inferred (for inference is all that is possible upon this point), the smaller tumor would appear to be the more rapidly growing of the two.

Diagnosis. Multiple hypernephroma of right kidney; slight chronic diffuse nephritis.

By the kindness of Professor Coplin, Dr. Ellis had the opportunity of examining a third specimen obtained post-mortem.

Case III.—The specimen was obtained at autopsy upon the body of a man aged forty-three, the pathological diagnosis being chronic adhesive pleuritis, arteriosclerosis, chronic interstitial nephritis, chronic interstitial pneumonia, hypernephroma of left kidney, arteriosclerotic softening of right cerebrum.

The left kidney was small, lobulated, and possessed an adherent capsule. On the convex border, just above the middle of the organ, was a slightly elevated, nearly circular nodule 1 cm. in its longest diameter. It was yellow in color and sharply differentiated from the surrounding tissue. The surface of the nodule was roughened by small projections, giving it a wart-like appearance. Incision showed the tumor to be almost spherical in outline and slightly more than 1 cm. in diameter.

Microscopic examination of sections from the specimen shows an encapsulated tumor situated immediately beneath the capsule of the kidney. The latter structure where it surmounts the tumor is considerably thickened and elevated. In addition to the general elevation, caused by the location of the growth, the capsule of the latter sends outward several projections

that produce irregular secondary elevations of the kidney capsule, and impart to it a papillomatous appearance.

The capsule of the tumor is a broad band of fibrous tissue, which, throughout the larger part of its extent, presents two fairly distinct but not sharply delimited zones. The outer is composed of rather dense tissue, containing but few bloodvessels and cells; the inner zone is not so dense, and shows more or less conspicuous invasion by tumor elements. This is particularly true of the part bordering the kidney where the evidence of extension of the growth is most marked. Here even the outer zone is extremely cellular, shading gradually into kidney substance undergoing progressive fibrous transformation. The larger bloodvessels in this area are the site of obliterative endarteritis. In this extending capsule, tumor elements are well advanced. At several points are quite large spaces better considered in connection with the tumor itself. Scattered throughout a large part of the capsule and extending into the stroma of the tumor, are granules and irregular collections of golden-brown pigment, both intracellular and extracellular in location.

The tumor proper is composed of a fibrous reticulum enclosing variously sized cell-lined spaces. The bands of the reticulum transmit capillaries and vary considerably in thickness; some consist of but little more than the endothelial wall of the capillary, others are distinct, though narrow, fibrous bands. The reticulum does not extend inward from the capsule as prominent narrowing and dividing trabeculæ, but, as stated in the description of the capsule, presents more the appearance of impinging upon and invading the inner portion of that structure. The cells lining the spaces of the tumor are large and usually polygonal in outline. They are attached perpendicularly to the reticulum, and where the latter is most scanty present the appearance of double rows or columns separated but slightly by the capillary wall. The nucleus is usually situated toward the centre of the cell and reacts well to chromatin stains; the cytoplasm stains very faintly, and in most of the cells contains large flat spaces. The spaces enclosed by the reticulum of the tumor vary in size from those essentially filled by the lining cells up to cavities 2 mm. in diameter. The smallest spaces contain detached cells of the type described and a few red blood cells; the large cavities are nearly filled with blood, and also contain desquamated cells in various stages of disintegration. A few of the better-preserved cells contain pigment similar to that described in connection with the capsule. The spaces previously mentioned as located in the capsule are similar to those just described, and are evidently extensions of the growth; the largest is 0.5 mm. in diameter.

The histology of that part of the kidney immediately bordering the tumor capsule has been described in connection with the latter structure. Farther from the tumor, and presumably uninfluenced by that body, the renal cortex shows a decided increase in connective tissue, with hyalo-fibrous

transformation of the glomeruli. The tubular epithelium is granular, fatty, and desquamating.

Diagnosis. Hypernephroma of left kidney; chronic diffuse nephritis.

SYMPTOMATOLOGY AND DIAGNOSIS, BY DR. PFAHLER.

As Dr. Keen has indicated, there is but one method of treatment, and that is operation; and the earlier it is performed the greater are the chances of complete recovery. All, then, depends upon an early diagnosis. With the object of working out the symptomatology and diagnostic factors, and at the suggestion of Dr. Keen, I have reviewed the literature upon the subject, have made an analysis of the clinical histories, and have tabulated the symptoms of the cases reported, including our two cases.

The great majority of the reports upon this subject contain only the description of the pathological nature and tendencies of these growths. In this collection I have included only undoubted cases of hypernephromas in which the clinical reports contained all of the symptoms and observations that were noticeable, but have not included the cases reported in which the symptoms were produced by metastatic growths. In very few were the clinical histories complete, nor were all the methods of investigation used that were at our command. Until all such methods of investigation have been used, we have no right to claim that a diagnosis cannot be made. I have collected and tabulated 26 cases. (See table.) The three most important symptoms or diagnostic factors connected with the disease clinically are hæmaturia, renal colic, and the detection of a tumor. All of these may occur together, any one may occur alone.

The most constant of these is *hæmaturia*, which is usually the first symptom complained of by the patient. In 63 cases studied from this point of view by Albarran,[6] it was the first symptom in 36. It occurred at some time during the course in 58 out of 77 cases, or in 75 per cent. Hæmaturia occurred alone in 4 cases, with a tumor in 28, with pain in 4, and with pain and tumor in 22. In the cases collected by me, hæmaturia was present in 19 out of the 26. It was associated with pain in 14, and with tumor in 15 cases.

It is probably only present when the kidney itself is involved, but, as Dr. Ellis will show, the kidney is involved in nearly all

CLINICAL ANALYSIS OF TWENTY-SIX CASES OF HYPERNEPHROMA, INCLUDING ONLY THOSE CASES IN WHICH THE CLINICAL HISTORIES WERE FAIRLY COMPLETE.

Author.	Reference.	No.	Pain.	Hematuria.	Tumor.				General symptoms.					Etiologic factors.				Special examination	Probable duration.	Result of operation.
					Location.	Size and shape.	Mobility.	Tender.	Emaciation, asthenia.	Color of skin.	Gastro-intestinal.	Blood.	Urine.	Age.	Sex.	Family history.	Traumatism.			
Morris.	Brit. Med. Jour., Lond., Vol. II, 1893.	1		At intervals during two years.	Right.				Marked.					43						Immediate result was good; died of metastasis.
Thorndike and Cunningham.	Boston Med. and Surg. Jour., Dec. 3, 1903.	2	Dull aching pain in lumbar region, and renal colic when hematuria ceased.	At intervals during three years; constant during six months; passed ureteral clots.	Right.	Convex, nodular.	Moves with respiration.	Tender.	"	Pale.	None.	Hemoglobin, 60%; whites, 6,500.	Sp. gr., 1,026; albumin, blood.	48	M.				8 yrs.	Successful.
"	"	3	First symptom, simulating renal colic, immediately after injury; later dull.	At intervals.	Right	Convex, smooth.	"	None.	Lost 25 pounds in 6 months.	Very pale.	"	Hemoglobin, 25%; reds, 2,770,000; whites, 6 200.	Sp. gr., 1,016; pus, no blood.	52	F.	Mother died of intestinal obstruct'n.	Followed traumatism to right lumbar region.	Cystoscopic examination showed clot in left ureter.	1-5 mos.	"
"	"	4	Dull aching pain in back was first symptom; pain occurred with and without hematuria.	Began one week after pain and lasted 48 hours; passed ureteral clots.	No tumor.	Abdomen symmetric.			Lost 10 pounds in 4 months; weak.			Whites, 12,000.		52	M.	Father, tumor of abdomen; mother, cancer of breast.			15 wks.	Died one week after; no cause determined.
Ohlmacher.	Cleveland Med. and Surg. Jour., March, 1903.	5	Renal pain, five years.	Recurrent hematuria during five years.	Right.		Movable.							19	F.				5 yrs.	Found at autopsy.
"	"	6		Periodic.					Edema of both legs.					52	M.				5 yrs.	Immediate result good; died of metastasis.
McWeeney.	Brit. Med. Jour., Feb. 8, 1896.	7	Abdominal pain.	None.	Left.	Size of a melon.	Fluctuation.		Delicate.		Vomiting; nightsweats			53	F.				9 mos.	Living four years after.
"	"	8	Abdominal pain.	"	Right.	Twice size of kidney.	Freely.		Emaciated.	Cachectic, pale.				38	F.				6 mos.	
Jellinek.	Cal. State Jour. of Med., Vol II, No. 2.	9	Recurrent renal colic, two years; first attack after running a race; dull pain five or six years.	Said to have occurred.	Right	Size of child's head			"	Slightly cachectic.			Albumin, pus and blood; indican, coagulation	43	M.	Negative.	Hematuria began in a faint at end of a race.		5 yrs.	Lived three months.
"	"	10	Pain in bladder, radiating toward penis	At intervals during two years.	Right.	Size of kidney.	Movable.	Tender.	45 pounds in 1 year; weak.	Pale.	Chills, fever and sweats.		Dendritic clots.	55	M			Cystoscope showed clot in right ureter.		Successful.
Boyd and McFarland	Proc. Path. Soc. of Phil., June, 1902.	11			Right			"					Negative		F.		Followed or associated with pregnancy.			Died.
Bierring and Albert.	Jour. Am. Med. Assn., July 23, 1901.	12	Renal colic recurred at intervals of three months and six months; then at intervals for two years and six months.	Began after six months, and recurred for two years and six months.	Left.		Fixed.							58	F.				3 yrs.	
Chandler	N. Y. Med. Jour., Sept. 3, 1901.	13	Renal colic developed after lifting a heavy barrel; recurrent during four years.	Recurrent with colic.	Right.		Movable.		Emaciation and debility.	Pale and muddy.	Gastrointestinal catarrh.			13	F.	Negative.	Followed the lifting of a barrel.		4 yrs.	Successful.
Lubarsch.	Virchow's Arch., Vol. cxxxv, 1894.	14		First symptom, recurred at intervals during six months.	Right.	Size of child's head.	Moves with respiration.							52	M.		No apparent cause.	Examination of bladder and kidney under ether, 3 months previous, gave increased resistance over the kidney.	6 mos.	Lived one day.
"	"	15	Renal colic, one year before hematuria, was the first symptom; recurrent until operation, one year later.	Occurred repeatedly beginning about a year after first attack of renal colic.	Right.	Size of child's head.	Movable.		Well nourished.				Acid, cloudy but not bloody.	64	F.				1 yr.	Lived one week.
"	"	16	None.	None.	Right.		Movable with respiration.						Negative.	73	M.				Over 8 mos.	No operation; metastasis developed after influenza.
"	"	17	Pain was the first symptom, in the region of the kidney, three years before other symptoms.	Bleeding began suddenly and without cause, and again two months later, after exertion.	Left.	Small.							Blood in urine.	50	M.	Negative.	An attack of bleeding occurred after exertion.	Cystoscope showed blood from left ureter; under ether, induration of kidney.	3 yrs.	Lived one day.
Plummer.	Ann. of Surg., No. 39, 1904, p. 468.	18		Only one hemorrhage fourteen months before operation; none again; pure blood.					Emaciation and weakness.	Pale.				68	M.				14 mos.	Operated upon.
Bevan.	"	19	Renal pain.	Hemorrhage.		Large kidney mass.			Secondary.	Anemia.	Diarrhea develop'd after operation.		No tubercle bacilli.					Röntgen ray examination showed no stone; no tubercle bacilli in urine.		Metastasis developed in the intestines.
Brown.	Ann. of Surg., No. 39, 1904, p. 299.	20	Renal colic three years before operation; recurred at times with the hematuria.	Hematuria three years before operation; recurred with and without pain.	Right.	Large nodular.		Early tender.	Marked; cachexia at time of operation.	Cachectic.			"	46	M.			Röntgen ray examination, no stone; no urine from right ureter 2 years before operation.	2 yrs.	Recovered.
Morris.	Brit. Med. Jour., Nov. 11, 1899.	21		Hematuria two years before operation, and only occurred once.	Left	Noticed six months before operation.	Freely movable.		Emaciation and debility developed after operation.	Pale.					M.			Varicocele on left side, found to be due to obstruction of the spermatic vein.	2 yrs.	
"	"	22	Renal pain.	Hematuria two years.	Right.	Tumor of right temple; palpable.			Emaciated.					43	M.				2 yrs.	Living eight months after; liver had increased in size.
Robson.	Brit. Med. Jour., Oct. 21, 1899.	23	Pain in region of gallbladder, radiating to the right shoulder; lasted two years.	No urinary trouble.	Right.	Noticed three weeks before operation.	Freely movable.	Not tender.	Asthenia; depression; emaciation				Negative.	47	F.			Colon inflated; tumor pushed up toward the liver.	2 yrs.	Lived one month.
"	"	24	Pain in right lumbar region, extending to the shoulder; about one year.		Right.	Palpable.			Emaciation; depression; asthenia.		Indigestion and flatulency.			57	F.				1 yr.	Living two years after.
Keen, Plaider and Ellis.	The present paper.	25	Vague pains in the abdomen.	None.	Right.	Twice size of kidney.	Movable.	Slight.	Emaciation and debility.	Very pale.	Nausea, vomiting, diarrhea, constipation.	Hemoglobin, 41; reds, 4,200,000; whites, 10,000.	Negative.	54	F.	Father, cancer of face; mother, cancer of breast.	Followed traumatism.	Röntgen ray showed dense areas in general shadow of the tumor.	1 yr.	Well eleven months after.
"	"	26	First symptom, renal colic at intervals for three years.	Hematuria at intervals during three years.	Right.	12 cm. in diameter.			"	"			Albumin, $\frac{1}{10}$; no casts, no tubercle bacilli.	52	M.				3 yrs.	Well four years.

cases, and therefore this distinction is of little practical importance. Usually the hemorrhage begins without apparent cause, and at times while the patient sleeps; but in three cases it seemed to be excited by traumatism or exertion. In one case it was caused by direct traumatism to the lumbar region; in another by lifting a heavy barrel; and in a third case it occurred at the end of a race, associated with fainting. In one of our own cases (Case II.) trauma may have been a factor in the causation of the disease. The blood is usually sufficient in amount to form clots, and may amount to a pint or more, and it may be passed as almost pure blood. Ureteral clots are often present.

Hæmaturia may be almost constant for several months and then be entirely absent for several months. It usually occurs at intervals, and lasts but a short time. Plummer[7] mentions a case in which hæmaturia occurred only once, fourteen months before operation. Morris[8] also reports a case in which hæmaturia only occurred once, two years before the operation. In seven of the 26 cases there had been no hemorrhage during the entire course, and no urinary symptoms. This was true in one of our cases (Case II.), and in this case half of the kidney structure was involved by the growth. Therefore the absence of hæmaturia does not indicate that the kidney itself is free from growth.

Pain. The usual form of pain is that which is commonly described as renal colic; but it may be a mere dragging sensation, or dull pain in the lumbar region, such as occurred in the cases of Thorndyke and Cunningham.[9] Morris mentions two cases in which the pain occurred in the region of the gall-bladder and the lumbar region, respectively, and radiated toward the right shoulder.

The pain usually occurs with the hæmaturia, and in such instances may be due to the formation and expulsion of clots from the ureter. It may occur without hæmaturia, such as was noted in one of Bierring's[3] and two of Lubarsch's[10] cases. In the study of this point by Albarran, pain was the first symptom in 20 out of 63 cases. Of these 20 cases, it occurred alone in 14, with blood in 5, and with tumor in 1. In 77 cases pain was present at some time during the course in 32. In my series it was present in 19 out of the 26 cases.

Tumor. A tumor may be discovered by the patient before any other symptoms have been produced. It was the first symptom in 12 out of 63 cases studied by Albarran. The same author found it present at some time during the course in 69 out of 77 cases, or in 89 per cent. In my series of 26 cases it was detected in 21. As a rule the tumor is detected after other symptoms have become manifest, and the interval between the earliest symptoms and the discovery of the tumor may be several years. In size the tumor varies, but may reach that of a child's head. Out of the 26 cases 4 were so described. The tumor was movable in 12 out of the 26 cases. This mobility varies from a slight movement with respiration to very free movement, and this forms a very valuable point in the differential diagnosis. Tenderness was elicited in 6 out of the 26 cases.

Location. The tumor was found upon the right side in 18 of the 26 cases.

General Symptoms. The general symptoms consist chiefly of a marked asthenia, emaciation, and languor or depression. These symptoms were present in 18 of the 26 cases. The color of the skin is usually described as being pale. In only two was it described as cachectic, and in none of them was it found to be bronzed. Gastrointestinal symptoms were complained of in 5 cases. In one of our cases (Case II.) this was the chief complaint.

The *blood* has been examined in only four cases, and in one of these only the leukocyte count was made. In the 4 cases the leukocytes varied from 6000 to 12,000. The hæmoglobin was comparatively low, once as low as 25 per cent. In one of our cases it was 41 per cent., while the erythrocytes were 4,200,000, the leukocytes were 12,000, and the differential count was normal. We had therefore in this case the blood picture of chlorosis.

The examination of the *urine* was reported in 13 of the cases, but, except when it contained blood, it was practically normal.

The *age* was given in 22 cases. The oldest patient was seventy-three, and the youngest thirty-eight, the average being fifty-two. Hypernephroma, therefore, as a rule, appears to be a tumor belonging to middle and advanced age. Albarran says it is rare before forty and after sixty.

The *sex* was mentioned in 24 of the 26 cases. It was found 13 times in the male, and 11 times in the female. This point was studied by Lubarsch in 28 cases, 19 of which were in the male. This disease therefore seems to occur rather more frequently in the male.

Heredity. The family history was mentioned in only 6 of the 26 cases. In 3 of these 6 there was a distinct history of carcinoma in the parents.

Duration. The duration of the symptoms varied from fifteen weeks to eight years, or on an average of two years and three months.

Grawitz and Strübing[2] report a case in which a tumor was present in the region of the kidney five years, J. Israel[11] one eight years, and Beneke[12] one ten years, while Askanazy[13] reports a case in which the tumor was discovered early in youth, then lay dormant for over twenty years, and produced symptoms at the age of fifty-three.

Hypernephroma therefore may be a slowly growing tumor. This should not, however, become an excuse for delay in operation, because at any time an intercurrent affection may cause rapid progress of the disease, followed by metastases. This was noted in two cases after an attack of influenza. One of these cases was reported by Lubarsch.

Special examinations were made in 9 cases. A cystoscopic examination was made in 3 cases, and in each showed blood coming from the ureter of the diseased side.

A Roentgen-ray examination was made in 3 cases. In 2 it excluded stone. In one of our cases, a shadow of the tumor and kidney was shown, and in the general outline more dense shadows were obtained which corresponded to the nodules of the tumor. In a case reported by Lubarsch, an examination under ether, which was made three months before a tumor could be felt, gave increased resistance over the kidney. In one of Robson's[14] cases the colon was inflated, and the tumor was pushed up toward the liver. In a case reported by Morris, a varicocele had developed upon the affected side, which was found to be due to a distortion and dilatation of the spermatic vein as it passed over the tumor.

Diagnosis. The diagnosis of these tumors is, of course, very difficult, since we have no pathognomonic sign or symptom, but I believe that it can be made in some cases by a careful consideration of the symptoms, coupled with a complete examination. The diagnosis simply of tumor of the kidney was made in a number of the cases reported, but it is most desirable that a correct diagnosis should be reached before any palpable tumor exists, and that an exploratory operation should immediately follow the diagnosis.

The diagnosis can only be made by the method of exclusion. Time and space will not permit of a review of the methods that may be used, but a study of the foregoing table will give an idea of the points to be investigated directly.

Having excluded the other causes of hæmaturia and of renal colic, and having detected or suspected a tumor of the kidney in a patient in advanced life, especially if it occur upon the right side in a male and is freely movable, we are justified in making a diagnosis of hypernephroma and in advising an exploratory operation.

PATHOLOGY, BY DR. ELLIS.

The terminology of tumors of the adrenal and the kidney is still in a state of considerable confusion. This is particularly true of certain adenomatous and lipomatous growths, regarding the histogenesis of which there exists a difference of opinion, and even with regard to carcinoma and sarcoma all authorities are not fully in accord. Grawitz, in 1883, to the views then held, introduced a conflicting element by his assertion that a group of the smaller tumors of the kidney, currently recognized under the name of lipoma, were in reality derived from aberrant adrenal glands. For them he suggested the name of *strumæ lipomatodes aberranti renis.* New interest in the subject was immediately aroused and the results of numerous investigations were soon published. In general, the views of Grawitz were supported, though dissenting opinions were by no means few in number. This has remained the status of the question up to the present time, although the opponents of the views of Grawitz have steadily lost ground as carefully studied cases have increased in number. To these tumors Birch-Hirschfeld, in 1896, applied the name of hypernephroma, a term now most often em-

ployed, though by some they are designated simply as adrenal tumors.

Histogenesis. It is not my purpose at this time to enter into a lengthy discussion of the histogenesis of these tumors. Those who wish to investigate this phase of the subject are referred to the admirable work of Kelly[15] for a critical summary of the earlier writings as well as the findings of careful original studies. Among the most extensive recent contributions are those in the works of Hoche and Albarran and Imbert. It should be said, as previously intimated, that the literature of the subject, since Kelly's paper appeared in 1898, is principally in the nature of reports of additional cases, lending weight to the original views of Grawitz. Apparently conflicting opinions are, for the most part, due to differences in histological interpretation rather than dissent from the original proposition. Hoche,[16] after carefully reviewing the subject, is unwilling to admit that the theory of Grawitz is absolutely demonstrated, but says it is sufficiently established for one to believe, until proved to the contrary, that certain intrarenal tumors originate in adrenal tissue.

Albarran and Imbert[6] regard the reasons given by various writers and summarized by them, as being fully sufficient to establish hypernephroma on a solid basis. They are convinced, however, that many tumors of the kidney, if examined by different pathologists, would be pronounced by some "epitheliomas with clear cells" and by other hypernephromas. This is due to the probability that malignant tumors with clear cells develop from aberrant adrenals, and also that neoplasms identical in appearance may take origin in the epithelium of the kidney. The future must teach us to distinguish between the two. Albarran and Imbert recognize three varieties of lipoma or pseudolipoma of the kidney: (1) The true lipoma described by Virchow; (2) pseudolipoma or hypernephroma of Grawitz; (3) pseudolipoma of Ulrich, resulting from fatty degeneration of the epithelium lining the convoluted tubules. Tumors of the kidney in general are placed by these authors in four groups: Group 1. Tubular and papillary adenomas and carcinoid epitheliomas with the transition form of adenocarcinoma. Group 2. On the one side the benign pseudolipoma of Grawitz and alveolar adenomas;

on the other side malignant hypernephromas, which include in great part the epitheliomas of clear cells. All these, with evolution benign or malignant, may come from capsular debris. It is also possible that a certain number of cancers with clear cells may originate in renal canaliculi. Group 3. Sarcomas, lipomas, and true angiosarcomas that originate from the mesodermic tissue of the kidney. Group 4. Mixed tumors originating in embryonal germs. They believe that the tumors described by Paoli, Driessen, and others under the name of endothelioma, angiosarcoma, etc., relate in part to carcinomas of clear cells, particularly to those which take their origin in aberrant adrenals.

Kelynack[17] holds that but few pathologists now deny the adrenal origin of certain renal growths. McWilliams[18] says "the pathogenesis of adenomas of the kidney is obscure. Some are undoubtedly of accessory adrenal origin, others arise from remains of the Wolffian body, others from isolated elements due to errors in development." Williams[19] thinks "this (suprarenal) origin for certain renal tumors may be considered as firmly established as any fact in renal neoplastic pathogeny."

Kelly,[20] in a later article, says that most of the so-called primary carcinomas and alveolar sarcomas of the kidney are in reality hypernephromas. Bland-Sutton[21] speaks of renal "sarcomas" arising in adrenal rests, and states there are no facts to support the assumption of some writers that renal sarcomas arise in the adrenal gland and gradually become incorporated with adjacent parts of the kidney. Morris[22] observes that the microscopic characters of this group of tumors are capable of various interpretations, and what one person classifies as typical renal tumor of accessory adrenal origin, another calls spheroidal-celled carcinoma, another endothelioma, still others epithelioma of clear cells, renal adenoma, primary angiosarcoma, alveolar sarcoma, or lymphadenoma. Norman[23] describes an encapsulated tumor an inch in diameter, half of which was within, the other half projecting from the surface of the kidney. The cells were cylindrical and loaded with fat. He believes there is not sufficient evidence to support the theory of Grawitz, and prefers the explanation of Sabourin, that these tumors originate in proliferated epithelium of the convoluted tubules of the kidney.

Parodi[24] grafted adrenals from embryo rabbits into the kidney, liver, and sciatic nerve of rabbits three or four months old and also into adult animals. The cortex became adherent, the medulla did not; after a time this was smothered by developing connective tissue. The morphology of the grafts in the kidney and liver led Parodi to the conclusion that adenomas of those organs assuming hyperplasia must be considered as adrenal inclusions. Neusser[25] says that excessive proliferation of circumscribed portions of suprarenal substance gives rise to small tumors resembling lipomas which have been termed suprarenal strumas or adenomas. They are situated in the cortex of the suprarenal capsule or more frequently in accessory glands occurring in the kidney. According to the latest views he believes they are to be considered as sarcomas of the vascular perithelium (perithelioma). Ricker,[26] in certain cases in which the adrenal was abnormally near the kidney, found renal tumors in the adrenal and adrenal tumors in the kidney. Of non-malignant tumors of the kidney he describes: 1. Tubular adenomas. 2. Trabecular cystomas. 3. Those of adrenal origin.

These references might be many times multiplied, but they are sufficient to demonstrate two things: 1. The majority of writers recognize the adrenal origin of certain tumors called hypernephromas found in the kidney and elsewhere at points near to, or remote from, the adrenal gland itself. 2. The difficulty of deciding in all instances between hypernephroma and adenoma, lipoma, or even carcinoma of either kidney or adrenal; this may be assigned as one reason for the existing differences of opinion regarding renal growths in general, and the one we are at present considering in particular. Hypernephroma, then, may be defined as a tumor arising from adrenal tissue, whether in the normally situated gland or in ectopic fragments of that organ known as adrenal rests. By common usage the term is seldom applied to growths of the adrenal itself, being reserved almost exclusively for those springing from aberrant particles of the gland. Primary hypernephroma, therefore, while usually in the kidney (96 per cent. of reported cases) is possible in widely different situations.

Embryology. The embryology of the adrenal gland throws much light upon this frequent occurrence of ectopia of the organ. Radasch[27] mentions that at one time in the develepment of the embryo the

adrenals are comparatively large and enclose nearly the whole of the kidney. As the fetal kidney is lobulated, he says it may be readily understood how small detachments of adrenal cells could, at that time, be enclosed in the interlobular connective tissue of the kidney. The development of the adrenal from the same type of tissue and in relation with the sexual glands accounts for the frequency with which fragments of the former are strewn along the paths of the latter in their descent. Radasch also states that ectopic adrenals occur in both sexes, the usual location being some point between the kidney and the descended sexual gland. They have been found in or upon the adrenal, kidney, and liver, in the perirenal tissue, solar and renal plexuses, mesentery, in the region of the internal abdominal ring, in the inguinal canal, upon the spermatic cord, between the epididymis and testis, in the broad ligament, and the fundus of the uterus. Marchetti[28] has also found them embedded in a serous cyst of a human ovary and in the ovaries of guinea pigs. The frequency of ectopic adrenals is placed by some writers surprisingly high. Imbert[29] says they are found in 92 per cent. of all bodies coming to autopsy, and in the kidney in 6 per cent. to 8 per cent. of cases. Holmes[3] places the number at 90 per cent. of all autopsies, the bodies being found in various parts of the genito-urinary tract. Duckworth[30] mentions the case recorded by Pitt, in which there were four adrenals, two being on the anterior surfaces of normal kidneys, an inch from the upper end. Wynn[31] has observed a high frequency of supernumerary adrenals in cattle. McFarland[32] does not believe they are so frequent in man as in cattle. He has made a careful examination in more than 1000 autopsies and in them has never seen a distinct supernumerary adrenal. Kelly (*loc. cit.*) found an instance of union of the adrenal bodies with the kidneys and misplacement of portions of the former within the latter. The tendency of the adrenal itself to abnormal development is shown by Flint,[33] who speaks of finding islands of the cortical substance in the medulla, and vice versa. Also of the medullary substance extending from the central vein to the capsule and the cortex from the capsule to the central vein.

Frequency. Without endeavoring to make the search exhaustive, I have found references in the literature to 160 cases of hyper-

nephroma, recorded since 1890; to these the present series of three should be added. Nearly or quite a dozen known unreported cases are not included in this list. Albarran and Imbert give the number of tumors of the kidney reported from 1890 to 1902 as 588; of these, 529 were subjected to histological examination, and 90, or 17 per cent., shown to be hypernephromas. In addition to those called hypernephroma, many tumors of the adrenal are reported as sarcoma or carcinoma, with the statement that they correspond partially or entirely to the description by other writers of hypernephroma. Rolleston,[34] in mentioning 26 cases of primary tumor of the adrenal, says that nine may be called either malignant adenoma or carcinoma, the former being closely allied to hypernephroma. Amberg[35] reports a primary malignant tumor of both adrenal glands in a child of two months, with secondary involvement of the liver. Though both primary and secondary growths reproduced the medulla of the adrenal, Amberg considers the tumor impossible of classification on such basis, because the histogenesis of the adrenal is not definitely decided. He, therefore, says it may be morphologically classed as either a carcinoma or an alveolar sarcoma. From his description it would appear that this tumor might, with propriety, be called a hypernephroma. Amberg refers to Pepper's[36] six cases as being similar, and gives a lengthy discussion of primary tumors of the adrenal. Woolley[37] discusses at length adrenal tumors. He speaks of "malignant" adenomas, but does not mention hypernephroma or even aberrant adrenals. From these statements will be seen the difficulty of determining the exact number of cases of hypernephroma, and the reason why no attempt has been made in this paper so to do. I am fully convinced the number cited is far too small, but cases reported as adenoma, carcinoma, or sarcoma must be accepted as such, even though their metastases produce what seems to be adrenal tissue.

That any attempt to number the cases must result in only an approximation, is recognized by Albarran and Imbert, who furnish elaborate tables of statistics regarding different tumors of the kidney, as to their frequency, symptoms, malignancy, etc., and then say there are so many bases of diagnosis that their figures are given only as curiosities.

Site. Of the 163 cases of hypernephroma included in this paper, 157 cases were in the kidney, 3 in the adrenal, 2 in the liver, and 1 in the uterus. This great preponderance of cases in one organ explains why the term "hypernephroma of the kidney" is almost invariably used in reference to this tumor. As previously stated, it appears as if the number in the adrenal might be greatly increased were it not for the tendency among observers to avoid this term for tumors of the adrenals themselves. Clinically, the problem would be simplified, and but little change in the classification made necessary, if the name hypernephroma were restricted by all pathologists to primary tumors, and their metastases, originating in aberrant adrenal tissue only, and not to those springing from the normally placed organ. Still better, in the opinion of the writer, would be the application of the term "adrenal tumor" to all new-growths producing adrenal tissue, regardless of situation. Clinically, of course, they would be benign or malignant.

Morbid Anatomy. In the kidney, hypernephromas are usually single, but not rarely multiple, and have been found in both organs. Regarding the side, they occur indiscriminately. Of the cases in this series in which it is specified, the tumor was in the left kidney 57 and in the right 55 times. As to sex, 71 were in males, 45 in females. The tumors are situated beneath the capsule of the kidney and vary in size from a pinhead to that of a pea, in what may be called the strictly benign growths, and from this to the size of a child's head in the frankly malignant cases. When small they are almost always confined to the renal cortex, though they may be in the medulla. When large they project from the surface of the organ and also extend inward at the expense of the renal structure until they reach the pelvis, which may be obliterated by pressure, but is seldom actually penetrated. Either pole or the middle of the kidney may be involved. In the former case one extremity, and in the latter both, may retain the renal outline and structure. The external surface of the tumor is lobulated by depressed bands of the capsule. The color is usually grayish-red or yellow, the latter predominating, but often there are brown or bluish or even black areas, due to the presence in the tumors of small or massive hemorrhages. The tumor may be firm, but in many of the larger growths the projecting masses

are softened, in some instances being almost cyst-like in consistency. Incision of the tumor reveals surfaces corresponding closely to the external appearance in color and in lobulation. Masses within alveoli formed by the fibrous stroma may be so soft as to project, and even detach themselves from the surrounding tissue. The tumor is generally sharply outlined from any remaining renal structure by a distinct band of firm fibrous tissue. Hemorrhages into the tumors are exceedingly common and areas of softening, due to degenerate changes, are also frequently present.

Targett[38] found in one of his cases, on the lower and outer surface of the kidney, a yellowish body the size and shape of a finger-nail. The capsule was split and through the lower part the adrenal tissue extended into the kidney. Conversely a few convoluted tubules passed into the adrenal structure. His second case was one of a tumor two inches in diameter. Thorndike and Cunningham[9] report three cases. They say the outline of the tumor is usually that of the kidney. It is commonly either encapsulated within the kidney or is situated immediately beneath the renal capsule. Eurich[39] found congenital atrophy of the kidney, the upper pole of which was two inches from the adrenal. The intervening space was occupied by a spherical tumor one inch in diameter, purple in color and encapsulated; on the upper pole was the normal adrenal. The histology of the tumor was that of the adrenal medulla, and Eurich believes the tumor originated in the adrenal gland. Plummer[7] reports the case of a man aged sixty-eight years, who had a hypernephroma of one kidney eight inches in length; connected with it were three cysts, each containing two ounces of fluid. Boyd and McFarland[40] report a case appearing to follow pregnancy in a negress aged thirty years; autopsy showed collapse of the tumor, the larger part of which had become necrotic. Between the tumor and kidney was fibrous tissue belonging to the tumor and also additional connective tissue, resulting from atrophy of the parenchyma of the kidney. Bevan[41] reports a case in which the tumor projected into the pelvis of the kidney like a polypus. Two weeks after operation the patient suffered an attack of diarrhœa and pieces of hypernephroma were found in the feces. Bevan has seen seven or eight cases, and considers hypernephroma the most frequent malignant tumor of the kidney.

Caird[12] speaks of an enormous adrenal tumor that had engulfed the whole of a kidney. Brown[13] reports a large tumor of the right side, lobulated like the fetal kidney, and almost entirely composed of adrenal tissue. He also mentions three cases observed by other surgeons. Brown says certain facts regarding his case afford reasons for the query as to the possibility of a kidney being so entirely made up of adrenal tissue, even at a time when it is not appreciably enlarged as to be essentially functionless as an excretory organ. Of interest in this connection is the statement of von Frisch[41] that the cryoscopic point of the urine from a kidney in which a hypernephroma had caused the disappearance of the pyramids was normal, thus leading to the supposition that the kidney was functionally perfect.

Morris (*loc. cit.*) says the relation, or apparent relation, of the capsule to the various tumors in and around the kidney is not reliable as a guide to their morbid anatomy. "The isolation from the renal parenchyma of a tumor situated immediately beneath the renal capsule is far from being decisive in favor of the theory of Grawitz; neither is the argument that the capsule of the kidney seems to be continuous over the tumor a decisive proof that the tumor originated in the kidney and not outside of it." A primary tumor of the adrenal may stretch part of the kidney over it as a hood. There is equal reason to believe that as a primary tumor grows downward it may invade and cause destruction of the kidney tissue. Morris[8] later reports two primary tumors of the adrenal involving the kidney.

Thompson,[45] in speaking of liver abscess, says: "In disease of other organs I have only once met pus simulating that found in liver abscess. The case eventually proved to be a hypernephroma occupying the upper end of the right kidney. The symptoms simulated those of hepatic abscess, and an aspirating needle was thrust through the tenth intercostal space in the midaxillary line. At a depth of about two inches chocolate-colored fluid exactly like liver pus was withdrawn. It contained a few white flakes. No amœbæ were found. Operation revealed a normal liver and an enlarged kidney, the upper end being occupied by a cyst as large as a small orange. This was drained. A small piece removed from the wall showed the mass to be a hypernephroma." Harris[46] says when these tumors originate in the adrenal proper they are commonly

separated from the kidney by connective tissue, and however much the kidney may be attached to or flattened by the growth, the latter may usually be separated and removed, saving the kidney to the patient. Bean[47] reports the removal of a tumor supposed to be a cystic kidney. Examination revealed a hypernephroma 15 cm. by 16 cm. ($5\frac{3}{4}$ inches by $6\frac{1}{4}$ inches) in size. A. P. Ohlmacher[48] reports two cases of hypernephroma of the kidney. One was as large as a child's head, removed by operation, with recovery of the patient; the other the size of a hickory-nut, found at autopsy. Karewski[49] reports a case of multiple hypernephroma of the kidney in which suppuration had occurred. The appearance of the gross specimen was that of typical tuberculosis with multiple cavities. He knows of no other case of suppurating hypernephroma, and accounts for the complication in this instance by the general condition of the patient, who, although only aged twenty-seven years, had had cholera, pneumonia, purulent pleurisy, appendicitis with abscess (opened), and cholelithiasis (operated). Chandler[50] observed a case in which the entire kidney, with the exception of the capsule, was replaced by adrenal tissue, measuring 24 cm. by 13 cm. by 11 cm. ($9\frac{1}{2}$ inches by $5\frac{1}{4}$ inches by $4\frac{1}{2}$ inches). Rupprecht[51] found a hypernephroma of the right kidney in a child aged two and a half years. There was no recurrence one year after removal.

When occurring in the liver, primary hypernephroma presents much the same appearance as those situated in the kidney. That reported by Vecchi[52] was in the right lobe above the adrenal impression, definitely circumscribed and stated to be the size of a nut, from which we can infer but little as to the dimensions. It was intensely yellow, glistening, and distinctly lobulated by white bands, which, surrounding and penetrating it, caused bulging of the tissues between. Noyes[53] found a large tumor of the liver nearly identical with the structure of the adrenal, and attributes its origin to an aberrant gland. There was no primary growth in the adrenals. To these two cases three more might possibly be added, but I consider them better placed under the head of ectopic adrenals.

But one case of hypernephroma of the uterus appears to be on record. This was exhibited by Eastwood[54] and was a large malignant growth attached to the fundus, being separated from the uterine

tissue by a fibrous capsule. The zona glomerulosa of the adrenal was represented, the tumor showing typically the features of a malignant adrenal growth. Eastwood also showed two such tumors in connection with kidneys.

Metastases. A most interesting part of the morbid anatomy of hypernephromas is the occurrence of metastases. This at once raises the question of the clinical character of this group of tumors. From the descriptions already given it readily may be seen that the small hypernephromas given rise to no symptoms and found only at autopsy are as benign as the adrenal rests from which they spring. It is just as evident that the larger growths, which extensively destroy surrounding tissues by pressure or by actually breaking through the capsule, possess malignant attributes. This attains its highest degree in the formation of metastatic groups in other tissues of the body. These varying clinical manifestations of hypernephroma not inaptly have been compared to true adenomas of other tissues which later develop malignant characters. On this basis, all hypernephromas may be regarded as potentially malignant. Eisendrath[55] says the so-called Grawitz tumors are an intermediate class between benign and malignant groups, occurring in the circumscribed form as benign, or in a more diffuse manner as malignant growths. Neusser (*loc. cit.*) calls attention to the fact that apparently benignant hypernephromas are capable of giving rise to metastases larger than the primary growth. Metastases are most frequently found in the lungs, the liver, and in bones, but almost all other tissues of the body have been invaded. The discussion of this phase of the subject will, we believe, prove of most interest and value if the various points are taken up in connection with the exceedingly instructive series of reported cases.

Schultze[56] obtained post-mortem specimens including several organs. There was a tumor of the left adrenal, the organ being four times its normal size. The right kidney was eight times as large as normal, nearly all tumor; this growth extended through the renal vein into the vena cava as far as the liver. There were a number of isolated metastases in the visceral layer of the pleura about the bronchi and in the left kidney. The lower epiphysis and adjoining shaft of the right femur was invaded and increased four

times in size; absorption was marked, the bone being easily fractured and even cut through by a knife. In the discussion, Brooks said he had seen two cases, one of which showed metastases in the corpora cavernosa. Kelly (*loc. cit.*) reports four cases of hypernephroma of the kidney and one of the adrenal, four of which gave rise to metastases and the fifth invaded the renal vein and ureter. The tissues involved in the secondary growths were the peritoneum, vesical plexus and mucous membrane, vena cava and lungs.

Bierring and Albert[4] report five cases of hypernephroma of the kidney, three of which (collectively) produced metastases in the liver, lungs, intestine, and retroperitoneal lymph nodes. They discuss at length the method of metastasis, which they believe occurs most frequently through the blood by way of the renal vein. That extension through the lymphatics does occur, though rarely, they consider proved by their case with involvement of the retroperitoneal lymph nodes and also by the case of Le Count, which included invasion of the lymph nodes in the left inguinal region. They believe involvement of the liver by secondary growths is due in most cases to retrograde embolism through the vena cava and hepatic vein. In the discussion of this paper McCallum, of Baltimore, said a number of cases of hypernephroma occurring at the Johns Hopkins Hospital indicated that the most frequent metastasis was by way of the bloodvessels. He does not, however, regard the secondary growths in the liver as best explained by retrograde embolism. In one case the liver was almost filled by nodules, and it is difficult to believe they could have come from the vena cava directly. He believes the tumor cells passed through the lungs, although none appeared to lodge in those organs. Coplin suggested the possibility of hepatic metastases through the portal circulation rather than the lungs, aberrant vessels from the adrenal structure furnishing the mode of entrance to that system. A. P. Ohlmacher (*loc. cit.*) reports a case occurring in the service of Van Hook in which the tumor and kidney weighed 4725 gm. The vena cava contained a thrombus extending through the diaphragm and also into both iliac veins. The liver was filled with metastatic nodules which Ohlmacher attributes to retrograde embolism. Brown (*loc. cit.*) showed three specimens obtained by colleagues. One patient died

three years after operation, from metastases (location not given). In the discussion of this paper, Lilienthal said he had seen three cases. One man died from prompt recurrence and a woman operated on one year ago has a probable metastasis in the frontal bone. Hardou,[57] in reporting a case in which the right kidney was the site of a tumor, says that when hypernephromas are malignant it is to a less degree than are sarcomas.

Clairmount[5] found an egg-sized hypernephroma in the lymph nodes at the bifurcation of the trachea. The patient had been operated on ten years previously for a tumor of the right kidney. At that time the right pleura was opened and, it is believed, infected by the tumor cells, which were carried by the lymph stream and deposited in the nodes. The tumor in the mediastinum had grown slowly under unfavorable circumstances. Holmes (*loc. cit.*) found in a subject, who had a tumor of the left kidney (or adrenal), the right lung filled with solid tumor masses and also three small foci in the left lung. Böhler[58] discusses 37 cases, 10 of which are original. Of these 13 had metastases, 12 in the lungs. The other sites of the metastases were in the opposite kidney, pleura, retroperitoneal lymph nodes, femoral vein, adrenal, femur, inferior vena cava, liver, dorsal vertebræ, ribs, temporal lobe and frontal sinus, and the wall of the left ventricle of the heart. J. C. Ohlmacher[59] reports the case of a woman of forty-nine from whose left kidney was removed a hypernephroma the size of an orange. She died in six weeks from metastases in the left temporal bone, right side of the neck, left breast and axilla, right abdominal wall, and beneath the line of incision in the lumbar region. The renal vein was invaded. This tumor reproduced the medulla of the adrenal. Jellinek[60] reports two cases. In one a tumor of the right kidney was firmly attached to the under surface of the liver and sent a cone-shaped projection into the inferior vena cava. Le Count[67] found in the right kidney of a subject one hypernephroma the size of a hen's egg and several smaller ones. A retroperitoneal growth extended into the bony pelvis and through the first sacral vertebra, producing a tumor of the buttock with ulceration and gangrene. Metastatic growths were found in the left femur and left iliac lymph nodes and pathological fractures of the right ilium and left femur had occurred.

Histology. As the microscopic characters of the tumors reported in this paper have been described with some detail, and are fairly representative, more than brief reference to certain general features of the histology of hypernephroma is not at this point demanded. The tumors reproduce more or less perfectly the structure of the adrenal gland, usually one or more layers of the cortex, rarely the medulla, being represented. They possess a fibrous capsule, but this may be penetrated and even partially destroyed by rapidly growing tumors. The two prominent features are the stroma and the cells. The former may be composed of quite dense bands of fibrous tissue, but commonly consists of capillaries with walls of endothelial cells only. In the larger growths, both types are frequently represented, vascular fibrous bands dividing the tumor into large lobules or alveoli which in turn contain a capillary network. The relatively large cells of the tumor are round, polygonal, or even columnar in type: usually they rest on the stroma or the endothelium of the capillaries, but often become detached and lie free in the larger spaces. The cytoplasm is scanty and granular and contains numerous vacuoles marking the former site of dissolved fat. The nucleus stains deeply and a nucleolus is generally conspicuous. By some, considerable diagnostic value is placed upon the fact that the nucleus and nucleolus stain dissimilarly. Glycogen can usually be demonstrated in these cells. Giant cells not infrequently occur. Kelly ascribes diagnostic importance to the presence of granules of intensely black pigment similar to those normally present in the adrenal cortex. He believes the cysts found in some hypernephromas originate in renal tubules enclosed within the adrenal tissue, and this point has been substantiated by other observers. In all tumors of any considerable size, degenerative changes are a prominent feature. Hemorrhages are often conspicuous, and are largely responsible for the marked color variations seen in many of the tumors.

Pathological Physiology and Chemistry. The question of increased arterial tension as a manifestation of hypernephroma may not improperly be considered at this point. This implies hypersecretion by adrenal tissue or the excessive introduction of its secretion into the circulation, and applies more properly to tumors

of the adrenal gland itself, but has been observed in cases of hypernephroma of the kidney. Certain writers strongly emphasize this point; the majority entirely disregard it. Ochsner[62] states that the tumors of Grawitz cause severe systemic disturbance, the principal symptom being greatly increased arterial pressure. "The patient rapidly becomes sick and dies, usually with a hemorrhage into the brain or other important structure." Sippy (*ibid.*) said he knows of only four reported cases in which adrenal tumor of the kidney was accompanied by greatly increased arterial tension. Two cases, seen post-mortem in Vienna, which were accompanied by cerebral hemorrhage sufficient to cause death, had high arterial tension during life. He states that Neusser and Holmes each report a similar case, but gives no reference to their papers. Sippy does not consider increased arterial tension a diagnostic sign of much value. An editorial writer[63] mentions two cases of high arterial tension accompanying hypernephroma. One patient suffered from persistent headache until the tumor was removed. The second was supposed to have renal sclerosis, the pulse being always of high tension, though the patient was pale. This writer says the size of the tumor does not determine the virulence of the intoxication. A small tumor may cause cerebral hemorrhage, or a large one may give rise to no marked circulatory symptoms. Vaquez[64] reports an adenoma of the adrenal removed at autopsy; the patient had had a persistently high arterial tension. The occurrence of secondary softening of the brain in Case III. of our series is exceedingly suggestive, even though the associated arteriosclerosis in itself might easily account for cerebral hemorrhage. Neusser (*loc. cit.*) suggests the possible diagnostic value of injecting into animals the urine of patients supposed to possess an adrenal tumor, to determine if it causes increased blood pressure. Cooper[65] says the technique necessary for this is too complicated for general use, and recommends injecting the urine into the ear of a rabbit and noting if changes occur in the easily observed vessels of that organ. The whole question of increased arterial tension in hypernephroma can be considered as entirely unsettled, clinical observation on this point having been sadly neglected. This is readily explained by the fact that the tumor is seldom diagnosticated before operation. The point is one

that should be investigated, however, in every case of suspected adrenal or renal tumor.

The diagnosis of gross specimens of hypernephroma by the iodine-starch reaction of Croftan[66] deserves mention. Several observers attest its value, but at present I cannot similarly endorse it. The reaction was obtained with a part of the tumor in Case II., after it had been for several months in formalin. An obscure tumor of the liver, thought possibly to be a hypernephroma, also gave the reaction. Testing its value further, we obtained a positive result with a tuberculous mamma, fresh, and a formalin-preserved mixed tumor of the parotid gland. Further experiments will be necessary to convince us of the specificity of the reaction of this test.

Since the demonstration that a part of the fat content of the cells, particularly the spongiocytes, of the adrenal gland is lecithin, the search for the substance in hypernephromas, as a diagnostic aid, has been suggested. The difficulty of isolating this substance rendered the question for some time of academic importance only, but Bernard, Bigart, and Labbé[67] appear, in part at least, to have solved that problem. Diagnostic value of lecithin in tumors, however, remains to be determined. Albarran and Imbert do not consider it any more than fat or glycogen, pathognomonic of hypernephroma.

The privilege of studying two of the present cases I owe to Dr. Keen; the third to Dr. W. M. L. Coplin. I am deeply indebted to both for their kindly interest and assistance in the preparation of this paper.

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DISCUSSION.

Dr. Charles P. Noble: I have had the experience of having one case of hypernephroma. The patient was a young married woman, who after giving birth to a child was found to have a tumor in the abdomen which the family physician looked upon as an ovarian cyst. The patient was referred to me with that diagnosis. It was evident upon careful examination that it was a case of tumor of the kidney and not an ovarian tumor. I have a drawing here which was made at the time. There was nothing special in the clinical side of the case. The patient was treated by removing the kidney, including the tumor. The tumor itself, as alluded to in the paper, was full of hemorrhagic areas. The patient made a good recovery and has had no trouble since.

Dr. A. O. J. Kelly: There are several aspects of hypernephromas to which a hurried reference may be made. First, as regards their frequency, my conviction being that they are by far the most common tumor of the kidney in the adult. I believe that one is almost warranted in saying offhand that a given tumor of the kidney in the adult is a hypernephroma; that is, about 90 per cent. of kidney tumors in the adult are hypernephromas. Without having figures at hand, I should say that I have studied between thirty-five and forty tumors of the kidney, and of these surely thirty were hypernephromas. During the last four or five years, at the German Hospital, I believe that we had a half-dozen or more. The question of the frequency of these tumors is very much bound up with that of the frequency of aberrant adrenal rests. As Dr. Keen has stated, they are quite common; in fact, they can readily be found in a large majority of bodies by anyone taking the trouble to search for them. Indeed, I have come to look upon such aberrant adrenal rests, as well as hypernephromas, as rather commonplace. A word with reference to the hæmaturia, to the presence of hæmaturia in some of the cases mentioned in the paper and its absence in others. This finds its ready explanation in the anatomical features of the tumor: In its early stages, in some cases for a rather long time, the hypernephroma is entirely surrounded by a connective-tissue capsule; as far as the kidney itself is concerned, the tumor, if I may so express it, is extrarenal, although a considerable portion of the kidney may be invaded. Thus, no opportunity is afforded for blood to gain access to the proper kidney structures and hæmaturia does not occur. Sooner or later, however, the tumor invades and finally penetrates the capsule, and hæmaturia results. In some cases the capsule may be imperfect, or invasion may occur early—in which cases hæmaturia also occurs early. As regards the diagnosis of hypernephroma—this is really the diagnosis of a tumor of the kidney, and as I have said, the recognition of a tumor of the

kidney in an adult is in the great majority of cases *prima facie* evidence of a hypernephroma. In addition to the symptoms detailed by Dr. Keen, I may say that some writer, whose name escapes me at the preseut time, has suggested that we might be led to suspect the presence of these tumors by auscultation and eliciting a souffle somewhat similar to the uterine souffle in pregnancy—this being occasioned by the great vascularity of the tumor. Seven years ago, when I published my first paper on tumors of the kidney, I had the temerity to offer the suggestion to surgeons, a suggestion mentioned by Dr. Keen to-night, that in operating on these tumors they should be careful to ligate the vessels before mauipulating the growth very much—this because of the likelihood of manipulation causing dispersion of tumor elements throughout the circulatory stream, the common mode of metastasis. Finally, with regard to certain chemical tests for hypernephromas I believe the less said about them the better.

THE DIAGNOSIS OF TUBERCULOUS CAVITIES IN THE LUNG.[1]

By HERMAN B. ALLYN, M.D.

Dr. J. Kingston Fowler, in 1888, pointed out the portions of the lungs first to be affected in tuberculosis, and the line of march commonly taken by the disease. He believes that the primary site is an inch and a half below the summit of the lung, and rather nearer to its posterior and external borders. Lesions in this situation tend to spread backward, possibly from inhalation of the virus while the patient is lying down. This line of extension explains why early evidences of tuberculosis may be found in the supraspinous fossæ when the physical signs beneath the clavicles are of doubtful import. From this primary focus, which in front corresponds either to the supraclavicular fossa or to a spot immediately below the centre of the clavicle, the lesions often spread downward along the anterior aspect of the upper lobe, about three-fourths of an inch within its margin, frequently occurring in scattered nodules. A second and less usual site, he says, corresponds on the chest wall with the first and second interspaces below the outer third of the clavicle. The progress of the disease is downward, but it rarely penetrates the interlobar septum, only five times in 152 consecutive cases examined post-mortem by Ewart.

In the lower lobe the early deposits are about opposite the fifth dorsal spine, and along the interlobar septum, which is roughly marked by the vertebral border of the scapula when the hand is placed upon the spine of the opposite scapula and the elbow raised to the level of the shoulder. The opposite lung may be affected with symmetrical lesions of later occurrence, or disease may be found

[1] Read December 7, 1904.

close to the interlobar septum corresponding on the chest wall to the upper part of the axilla.

The portions of the lung first to be attacked are of importance in the present discussion, because cavities usually form first where the lesion is oldest. While Dr. Fowler's statement may be accepted as expressing the usual beginning and line of march of the disease, it should be mentioned that one sometimes finds the first evidences of the disease in the fringes of the lung which border the sternum, in the first, second, and third interspaces, or high up in the axilla, or in the lappet of lung which covers the heart.

Ewart[1] gives the following figures showing the location of cavities:—

At the apices	282	instances.
In dorsoaxillary region	227	"
In mammary region	189	"
In sternal region	61	"
At base	32	"

Clinically one finds cavities most frequently anteriorly from the apex of the lung to the third interspace; posteriorly, in the supraspinous fossa, between the scapulæ and the spine, or beneath the scapula; next high up in the axilla, and, least frequently, at the base. Probably cavities must have reached the size of a walnut before they give distinct physical signs.

In the diagnosis of cavities from tuberculous disease of the lung the history of the patient may be of some service, especially bearing upon the duration of the disease and the quantity and character of the expectoration. While tuberculosis of the lungs spreads much more rapidly in some persons than in others, in the great majority of cases by the time cavities have formed the disease has lasted at least several months, and in many cases more than a year. It is especially in the slowly progressing chronic cases that cavities ultimately form. In acute phthisis and in the pneumonic forms death usually occurs before softening has progressed far enough to result in excavation. Moreover, when cavities exist, expectoration is more profuse, especially in the mornings, or after a change of posture, and is often nummular. I have frequently seen a patient with a

[1] Croonian Lectures, 1882.

cavity at the apex of the lung lie curled up on the affected side. This posture was evidently assumed in order to avoid the distressing cough caused by constant leakage from the cavity into the communicating bronchial tubes. Of course, after the cavity had become filled with pus, coughing would occur in spite of posture; but change of posture to the opposite side, or sitting up to take food, would result in hard spells of coughing, with profuse expectoration. In other cases in which the cavity is relatively dry, the patient may lie preferably on the opposite side. The cough is often exhausting, is followed by much dyspnœa, and the sputa are not so profuse.

But aside from the duration of the disease and the character of the expectoration, which after all can only excite suspicion as to the existence of a cavity, the statements of the patient are not of much value in diagnosis. Precise information can be gleaned from the physical signs alone.

Before speaking of the information furnished by the different methods of physical exploration, I wish to insist upon the importance of (1) a thorough examination, and (2) upon the importance of following an orderly progression in developing the physical signs, A thorough examination cannot be made without inspection of the chest, and oftentimes not without inspection of the entire chest uncovered. In women, from motives of delicacy, it is usually best not to bare the entire chest at one time; but even in them in every case in which inspection of the anterior portion of the chest alone leaves the condition obscure, inspection of the posterior and axillary portions can be carried out separately by arranging the clothing so as to cover the parts already examined.

By following an orderly progression, I mean that after the patient's history has been obtained by patient inquiry, examination should proceed by first obtaining all the information possible by a careful and systematic inspection of the chest, and that inspection should be followed by palpation, percussion, and auscultation in the order named. Most of the mistakes in diagnosis of chest conditions result from failure to follow patiently a good method, rather than from lack of knowledge; the examiner without looking at the chest, or with only a hasty glance, proceeds at once to percuss and auscult, and speedily reaches a conclusion, which may be correct, but is very

likely to be at least quite incomplete. The above method is the one I have endeavored to school myself into following. It has given good results. It is applicable to acute and chronic conditions of the lungs, and, for the matter of that, to the body in general. There is nothing novel in it unless it be insistence upon the importance of inspection.

Taking up the methods of examination then, in the prescribed order, the first is—

Inspection. It is surprising how much information can be obtained by a careful and systematic ocular examination of the chest. The first essential is a good light. If the patient be lying in bed with one side toward the window, the opposite side will be somewhat in shadow. This position may be unavoidable, but should be remembered in estimating the amount of expansion. When the patient is lying down the examiner must be careful to see that he is lying perfectly flat, without having a shoulder or hip raised above the level of its fellow. If this precaution be not taken no reliable results from inspection can be had. The best posture is a sitting one, with the face toward the source of light. The patient should sit erect, with the arms hanging loosely by his side. He must not lean to one side or the other. The general size and shape of the chest are not of much importance in the present discussion, because tuberculosis attacks all kinds of chests, and is, of course, more frequently found in normally shaped chests, because the normal type predominates. When tuberculosis has progressed to the stage of cavity formation there is always loss of flesh, and not infrequently extreme emaciation; sometimes as much as forty or fifty pounds have been lost before the patient is seen. As the result of this loss of flesh, the cheeks and temples are hollow, the neck thin, the ears prominent, the clavicles, ribs, and scapulæ appear to project, and there are depressions in the fossæ above and beneath the clavicles, and above the scapulæ. The fingers are often clubbed. Expansion is usually defective on both sides. To see a difference in expansion often requires close inspection under favorable conditions. The best posture for the patient is a sitting one with the light falling equally on the two sides, and the examiner either standing behind the patient and looking over his shoulders, or standing to

one side with the eye nearly on a level with the surface of the chest. It is true, one can often see deficient expansion when standing directly in front of the patient, but this is not the best position and results obtained by it are not to be relied on. Inspection should first be carried out while the patient is breathing quietly, and then he should be asked to take deeper breaths. The region from the nipple to the clavicle is the one where cavities are to be expected, and therefore it should receive particular attention. Deficient expansion in this region is highly significant of disease, for almost always the side that expands less is the diseased side, or is the more diseased if both are affected.[1] There will often be noticed, too, a little less fulness or roundness, some flattening in fact, where the expansion was diminished. This is often seen better in the second or third interspace toward the sternum rather than just beneath the clavicle. The second interspace often appears wider and deeper than its fellow of the other side. Moreover, the shoulder on the affected side often droops a little; and if the examiner stands behind the patient he will be struck by the lessened up and down movement of the shoulder, and lessened motion of the scapula. The supraspinous fossa of the more diseased side often shows distinct depression compared with its fellow.

Sometimes the entire side, anteriorly and posteriorly, shows defective expansion; generally the deficiency in expansion is limited to the apex. In some cases, also, expansion on the affected side may be noticed to be delayed, to lag behind, in point of time, that of the sound side. In only one out of twenty-four cases examined by Sibson with his chest measurer, was the expansion greater on the side having a cavity.

Wilson Fox says, apparently on the authority of Walshe, that occasionally a large cavity with thin walls may even cause a slight local bulging, and under these circumstances the expansion may even improve. Fox adds that in some cases also expansion is greater over a cavity than over a consolidation. This might be the

[1] Since writing the above I have seen a patient with deficient expansion of the left side and cavity at the right apex, the physical signs of which were especially marked in the supraspinous fossa. There possibly, in this case, had been an old pleurisy on the left side and then, after a considerable interval, disease began at the right apex and progressed rapidly.

case if in the consolidation the bronchial tubes were occluded, or the lung covered with a thickened pleura. I have seen bulging occur over a cavity while in the act of coughing; and when the cavity is near the anterior surface of the chest and covered with a very thin wall, the overlying interspace may be seen to flap in and out in respiration. Sibson mentions the fact that some slight recession may be occasionally observed at the commencement of inspiration.

Of course, it is well understood that obstruction of the bronchial tubes and feeble respiratory effort will diminish expansion in a cavity as in any chest condition. But there is rarely complete absence of motion, although the degree of motion is almost invariably less than on the other side, even though that, as is indeed very commonly the case, shows disease at the apex in the stage of consolidation.

Sibson[1] says: "Although the cavity has, over its centre, almost always an inspiratory movement, yet at its margins I have often found the motion abolished, and even reversed. The fourth costal cartilage is often over a consolidated portion of lung, which forms the walls of the cavity. The fourth costal cartilages receded either at the beginning or during the whole of an inspiration in fourteen out of twenty-two cases. The fourth cartilage receded in six out of ten cases on the right side, and in eight out of twelve cases on the left. Of thirty-nine cases of cavities in one lung there were eleven in which the upper end of the sternum fell in at the beginning of inspiration. In many cases both around and over the cavity the thoracic wall stands still just at the beginning of an inspiration. The lower end of the sternum, and the adjoining sixth cartilage on the affected side, recede, either at the beginning of inspiration, or throughout, in about one half the cases. Here the falling in is due to the elongation of the affected lung through the action of the diaphragm, and its consequent collapse." "If there be diminished motion during tranquil breathing, without any morbid cause, the difference in motion will usually disappear during a deep inspiration."

[1] On the Movements of Respiration in Disease and on the Use of a Chest Measurer. By Francis Sibson, Med.-Chir. Trans. vol. xxxi., London, 1848.

Sibson further says: "From these observations we may conclude that wherever and whenever an extensive cavity exists in the lung, the respiratory movements are restrained over that cavity, but not obliterated; that the respiratory movement is greater over the centre than over the circumference of the cavity, and that immediately over the circumference the ribs or sternum often recede, either during the whole inspiration, or, which is more usual, only at the beginning of it. The firm, tendinous, pleuritic adhesions that surround the lungs in the advanced state of tuberculous disease have more restraining influence over the movements than the disease itself has."

Palpation. Palpation is less valuable than inspection as a method of diagnosis in cavities in the lung; yet it gives useful information. When the light is defective or the patient lies in such a way that one side is in shadow, the examiner's hands may be applied to corresponding portions of the chest, and, when the patient is asked to take deep breaths, a difference in expansion is detected by the lessened motion which is then transmitted to one hand. This is a useful measure. Many persons will appreciate much better a motion that is felt than one which is only seen. The fremitus is generally distinctly increased when the cavity is empty, but if there be much fluid in the cavity it may be nearly absent. It is rarely as intense as that over consolidation. Sometimes there is very little difference in fremitus on the two sides, partly due to feeble vocal efforts by the patient, and partly to obstruction of the bronchial tubes leading to the cavity. If the patient have a chronic laryngitis no fremitus may be obtainable.

Percussion. Percussion must be practised with extreme care, and with the ears keenly alert for slight changes in sound, particularly in pitch and quality, or the results will be either negative or positively misleading. The note obtained over a superficial cavity sometimes resembles very closely normal pulmonary resonance. But on listening intently one discovers that it has less volume than the former, while it has a higher pitch and lacks vesicular quality. All resonance which is non-vesicular in quality Flint classes as tympanitic resonance. Hence the note I am describing would be classed as tympanitic; but it often lacks metallic or musical quality.

To my ear it is a muffled sound, without recognizable quality. It seems to be the same sound that West described as "boxy," and Musser and others have spoken of as "wooden tympany." This sound is not characteristic of cavity, however, for it may be heard in pneumothorax, and over the upper lobe of the lung in pneumonic consolidation, and above the level of a pleural effusion. In most cases, however, the percussion note over a cavity is either dull or appears so at first. I find that students set to examine a patient with a tuberculous cavity at the apex nearly always report that there is dulness or flatness on percussion. The reason for this is, that the percussion note is so high-pitched, so small in volume and short in duration, that it is indistinguishable from dulness, unless the quality of the sound is observed. The quality of the sound is tympanitic, that is to say, there is a slight musical or metallic intonation imparted to it. The tympanitic quality is usually faint, and may not be heard at all unless the patient be instructed to keep his mouth open while percussion is made. Forcible percussion is generally unnecessary, but the plexímeter finger should be applied firmly and struck a sudden, sharp blow. Flint advises that the examiner's ear should be brought into close proximity to the patient's open mouth during percussion; or, what is still better, that the pectoral end of a binaural stethoscope should be brought close to the patient's mouth, when the tympanitic sounds may be appreciated. It is only fair to say that even with these precautions no tympanitic or amphoric quality may be detected. In such cases either the cavity, if superficial, is filled with morbid, fluid contents, or the bronchial tube leading to it is stopped up, or the cavity is more remote from the surface and is surrounded by consolidated lung which gives its character to the percussion note. West thinks the percussion note is hardly ever tympanitic; but I am sure that when percussion is made with varying force, with the mouth open and closed, at the end of a held inspiration, and in varying postures of the patient, a tympanitic note will often be obtained. But I have found it present one day and absent the next, probably owing to changed conditions within the cavity, particularly its being filled with fluid contents sometimes and relatively empty at other times. This very changeability in percussion note is suggestive of a cavity.

Two varieties of tympanitic resonance, the cracked-metal or cracked-pot sound and amphoric resonance, are, as Flint says, quite distinctive of pulmonary cavity if found within a circumscribed space. But they are not pathognomonic. The cracked-pot sound may rarely be developed over consolidated lung, especially when the latter is overlaid with relaxed lung tissue. West says it may be found even over a pleuritic effusion. He says the best example of it he ever saw occurred over an enlarged heart when the lung was perfectly healthy.

The cracked-metal sound is developed over large-sized cavities, near the surface, and communicating with a patulous bronchial tube. It is best developed by fairly strong percussion, the striking fingers being allowed to linger longer on the pleximeter finger than in ordinary percussion; moreover, the patient's mouth should be open, and percussion should be made while he is inspiring.

Amphoric resonance is obtained over smooth-walled cavities which are empty and are surrounded by rigid non-collapsible walls.

Respiratory Change of Note. Friedreich has called attention to the heightened pitch of the percussion note during inspiration. It may cease to be tympanitic if the tension becomes very great. Wintrich's change of note consists in the note becoming higher in pitch when the mouth is open, and lower when the mouth is shut. According to Gerhardt's change of note, the pitch would be higher when the patient is sitting or standing than when he is lying down.

Auscultation. Auscultation furnishes us with some of our most characteristic signs of cavity. The voice sounds are usually transmitted with increased intensity; they may have a hollow sound, or exhibit amphoric quality. Flint declares that while the vibrations may be very intense they do not present the characters of bronchophony, namely, concentration, elevation of pitch, and nearness to the ear. These characters denote solidification of the lung. If the word pectoriloquy is limited to mean syllabic reproduction of sounds, so that the ear applied to the chest may hear not only the vibrations, but distinguish the syllables and words spoken, it must be a very rare sign. I have listened to a great many lungs and do not recollect that I ever heard it but once. I do not think most examiners make any distinction between the reproduction of

sounds with increased intensity on the one hand, and bronchophony and pectoriloquy on the other. Flint says: "Articulate words may be conducted by solidified lung as well as, if not better than, by the air in a cavity. There is, however, a cavernous pectoriloquy easily distinguished from that which denotes that solidified lung is the conducting medium. If the latter be the case, the pectoriloquy is associated with the characters of bronchophony; we may distinguish this as bronchophonic pectoriloquy. If the speech be transmitted solely through a cavity, the bronchophonic characters are wanting. Then the pectoriloquy is truly cavernous. This distinction I suppose to be original; I have for many years been accustomed to teach and illustrate it clinically."

Probably most persons use the word pectoriloquy to cover the reproduction of spoken sounds with increased intensity and nearness to the ear; and some add to this, that the sound has a hollow quality. But the actual hearing of articulate speech by applying the ear over a cavity must occur very rarely. In most instances I suspect that we know what the patient is saying and so think we can distinguish the words, or else we hear them through the air outside the chest.

It has seemed to me that the whispered voice sounds were oftener transmitted with increased intensity through a cavity than through solidified lung; though, of course, they may be heard through either medium. One would expect the whispered voice sounds to have lower pitch when heard over a cavity than when heard through solidified lung. Sometimes they are heard with rather startling clearness in contrast with ordinary speech and with the fremitus, which may have been defective. This so-called whispering pectoriloquy I regard as one of the most constant signs of cavity. In fact, the cavity may be mapped out by the area over which whispering pectoriloquy is heard.

The breath sounds over cavities are of much greater importance than the voice sounds. There are cavernous and amphoric breathing and rales.

Cavernous breathing is a low-pitched, blowing sound, without definite quality, with expiration lower pitched than inspiration, and both sounds variable in length. Its chief distinctions are its low pitch,

and the absence of vesicular, tubular, or amphoric quality. It is heard over superficial cavities with flaccid walls. In my experience this sound is rarely heard pure; almost always it is mixed with a tubular sound denoting consolidation, or has joined with it a faint amphoric quality. This admixture of sound is the natural result of the physical conditions which exist around the cavity. Often the cavity is surrounded on all sides by consolidated lung, which imparts a tubular quality and high pitch to the breath sounds. Sometimes the inspiratory sound is cavernous and the expiratory tubular, or vice versa. Or, again, there are often a number of small cavities, communicating or not, some of which have flaccid walls and some rigid; hence, we have a sound which is higher in pitch than pure cavernous breathing and of a faint amphoric quality, particularly on expiration.

When the cavity is large and its walls are rigid, amphoric breathing is heard. Flint says that an amphoric sound, if distinct, be it never so slight, always denotes pulmonary cavity, provided pneumothorax be excluded. The sound is analogous to that heard over a foot-ball when some one is forcing air into it after it is already full of air. It is a high-pitched sound of metallic quality.

Laennec has mentioned two cases of cavity in the lung in which metallic tinkling was heard in speaking and coughing; and Stokes,[1] who refers to this observation, himself has met with three cases. In all there were communicating cavities.

Osler refers to a curiously sharp, hissing sound as if the air were passing from a narrow opening into a wide space. When the cavity is very large and contains thin fluid a succussion sound may be obtained when the patient is abruptly shaken. In the rare cases in which a whole lung is excavated, leaving only a thin shell of lung or a thickened pleura, the coin sound may be heard.

The rales which are heard over a cavity that contains fluid are numerous, moist, and variable in size. Perhaps a listener is most impressed by hearing so many rales of a size, varying from large to small, but all, or nearly all, moist. One hears moist subcrepitant rales, mucous rales, bubbling and gurgling rales. They are often

[1] A Treatise on the Diagnosis and Treatment of Diseases of the Chest. Philadelphia. A. Waldie, 1837.

heard both in inspiration and in expiration. In addition there may be squeaking sounds, resembling sibilant rales, and rubbing sounds, which are probably pleural frictions. I have heard a high-pitched, clicking sound, simulating that heard in a telephone receiver on metallic circuit when the current is opened and closed. It was heard in inspiration and in expiration. But the lung sound is not quite so metallic as the telephone sound. Moreover, the various rales described may have amphoric quality, and even be accompanied by an amphoric echo. After coughing, the rales often become resonating. The heart sounds may be reproduced in the cavity and have amphoric quality, or blowing murmurs of cardiac origin may be heard.

Cardiopulmonary Signs. A cavity at the apex of the left lung may result in contraction and the drawing up of the heart, or sometimes its displacement outwards. Occasionally when this happens a systolic murmur is heard in the second and third left interspaces. The cause may be a kinking of the pulmonary artery. But, of course, there may be conjoined disease of the heart and a cavity in the lung. I have such a patient under observation now. There is a marked double mitral murmur and a systolic aortic murmur, and a cavity at the left apex. The aortic murmur was present before tuberculosis developed, and the heart is not displaced.

To sum up, our most trustworthy signs of tuberculous cavity in the lung are to be found in deficient expansion and flattening of the chest wall over the cavity; in a percussion note, which is often a high-pitched tympany, especially if percussion be made with the mouth open, but which may be only a muffled sound, or a dull or flat sound; in pectoriloquy (so called), particularly whispering pectoriloquy; in breath sounds which are cavernous, tubulocavernous, tubuloamphoric, or amphoric; and in a multiplicity of rales, chiefly moist rales, which after coughing may have resonating quality.

The following cases illustrate the conditions found in examining patients with tuberculous cavities in the lung:

A. H., black, aged forty-eight years, was admitted to the Philadelphia Hospital August 17, 1904. She complained of cough, dyspnœa, expectoration, loss of flesh and of strength, and night-sweats. The patient's father was dead of heart disease, her mother was living and well. One sister was living and

well, but another living sister had consumption. The patient had had the ordinary diseases of childhood, but had otherwise enjoyed comparatively good health. She had hemiplegia three years ago in this hospital. She admits moderate use of alcohol.

The general examination of the patient showed a much emaciated colored woman, markedly dyspnœic, with cough and much expectoration. The pupils were equal and responded quickly to light and accommodation. The tongue was moist, red, teeth marked along the edges. The pulses were equal, rapid, of moderate tension and compressible. The arteries were slightly thickened. Inspection of the chest showed marked emaciation, with deep depressions above and below the clavicles and conspicuous ribs. There was diminished expansion on both sides, but less on the right side, and some flattening of the upper portion of the right side anteriorly. On palpation vocal fremitus was increased on the right side. The percussion note on the right side was short and high pitched, with slight metallic quality, which did not change when the mouth was opened. Auscultation disclosed increase of vocal resonance. The whispered voice sounds were especially distinct. The breath sounds were obscured by an abundance of rales of all kinds, mostly large, moist or bubbling rales, which on coughing had resonating quality. The inspiratory sound when heard was short and high-pitched; the expiratory sound was lengthened, higher in pitch than the inspiratory, while both had amphoric quality. Posteriorly below the angle of the scapula the percussion note was dull instead of high-pitched tympanitic. The vocal resonance and fremitus over this area were increased, and the breath sounds tubular but obscured by a great number of sibilant and sonorous as well as mucous rales. Just above and immediately below the clavicle, tympany on percussion is very clear. The voice sounds have amphoric quality on the right side. Among the rales are numerous squeaking sounds which might have been pleural frictions.

On the left side the percussion note was hyperresonant, with slight dulness over the clavicle and in the suprascapular fossa. Anteriorally vocal resonance and fremitus are less than on the right side. Posteriorly the fremitus is a little more distinct on the left side. There was some tenderness on percussion on the left side. There was marked retraction of interspaces on the left side posteriorly, less in the right midaxillary line. The breath sounds on the left side show rough, rather short inspiration, and prolonged very faint but bronchial expiration. The prolonged expiration is almost cavernous beneath the left scapula posteriorly.

There was some œdema of hands, legs, and face; almost no change in finger-ends. The patient had a hectic temperature ranging from 97° to 102°, a pulse rate ranging from 85 to 130, while the respirations varied from 35 to 55 per minute. The urine was of low specific gravity, 1010, contained a small amount of albumin and some hyaline casts. The bowels were loose, the stools numbering two to four in twenty-four hours. The sputum contained tubercle bacilli.

The patient died September 11, 1904. An autopsy was held the following day. The pathological diagnosis was chronic bilateral adherent pleurisy; fibroid myocarditis; pulmonary tuberculosis with cavity formation; chronic diffuse nephritis; atheroma of the aorta; tuberculous ulceration of the intestines.

Passing over the other organs, the condition of which is sufficiently indicated in the pathological diagnosis, the state of the lungs was reported by the pathologist, Dr. Funke, as follows:

Left lung crepitates throughout, save lower portion of lower lobe, which is irregular in outline, measures four by six inches in diameter, is firm, cuts readily, section sinks in water. The cut surfaces are not refractive: scattered over them are large, gray points, 4 by 8 mm. in diameter, circumscribed, round or oval, denser at the periphery than at the centre and resembling caseation necrosis. The cut surfaces of the remainder of the lung are pinkish-gray in color with black markings and covered with pinkish frothy fluid. The right lung is large, bluish-black in color, does not crepitate, cuts readily, and cut surfaces are grayish-black. There is a cavity at the apex 4 by 10 cm. in diameter, containing a grayish-white substance (caseous). The walls are irregular and made up of caseous substance, and into this cavity many small similar ones open. The lower portion of the upper and the lower lobe has many cavities, varying from 4 mm. to 1 cm. in diameter, and of similar structure to that at the apex. There are also grayish-white circumscribed areas, 1 c.c. to 1.5 c.c. in diameter, firm at the periphery, soft at the centre, composed of substance simulating caseation.

The post-mortem findings in this case account very satisfactorily for the physical signs. The notes, however, do not state what was the fact, that the walls of the main cavity at the apex were firm.

W. E. L., white, aged sixty-two years, driver, was admitted August 21, 1904. He complained of cough, dyspnœa, expectoration, loss of flesh and strength, night-sweats and pain in the chest.

The patient's father had died of consumption, his mother of bronchitis; he had two sisters living and well, one brother killed in war, and one sister died of consumption. The patient had had the ordinary diseases of childhood, and gonorrhœa many times. He was given to excessive use of alcohol and tobacco.

He had been ailing for fifteen months. The pupils were equal, dilated, responded to light and distance. The tongue was dry, heavily coated, tremulous. The pulses were equal, of high tension, full, regular, compressible, the arteries thickened.

The patient was a much emaciated man, who said he had lost forty pounds. The clavicles were prominent, the scapulæ winged, the right shoulder was lower than the left. Posteriorly, the right side seemed shrunken. Right suprasternal fossa deeper than left and right lung anteriorly, especially at apex, expands less than left, and as compared with the left there is very slight flattening beneath the clavicles. Vocal fremitus and resonance are increased on the right side down to third rib, and the whispered voice sounds are reproduced with even greater clearness than the ordinary voice. Above the clavicle, over and beneath the clavicle, the percussion note is tympanitic, especially just beneath its middle portion. The musical quality comes out much better with mouth open. Pitch of the note is very high, almost if not quite as high as that of dulness or flatness. The breath sounds are amphoric down to third rib, anteriorly. After coughing there are high-pitched crackling rales somewhat resonating. Below third rib percussion note is somewhat hyperresonant. Inspiration is slightly bronchial. No change in vocal resonance or fremitus, and only occasional rales. Percussion note above left clavicle is dull, and slightly dull beneath left clavicle. The breath sounds were feeble, and after coughing on deep inspiration there were a few crackling rales.

The temperature was normal from admission except on August 23d and 24th, when it rose to 100°, and then fell afterwards to normal and continued so until death.

The sputum contained tubercle bacilli.

F. T., white, aged twenty-three years, a native of Japan; sailor. Admitted September 12, 1904. The patient complains of cough, dyspnœa, expectoration, loss of flesh and strength. His father and mother are living and well; two sisters are living and well; three brothers are dead, causes unobtainable; also past medical history unobtainable.

The patient is a poorly nourished young Japanese, who has been complaining for about a month and a half. The tongue is moist and slightly coated. The pulse is regular, of good volume, compressible.

When examined September 16th, the chest was moderately emaciated, the left clavicle more prominent than the right. Expansion was diminished over the left lung. Breathing was almost altogether abdominal. Fremitus increased on right, diminished on left side. The left apex and above clavicle, and to a less extent below, has percussion note impaired, high pitched with slight tympanitic quality beneath outer portion of clavicle. From second rib down percussion note is hyperresonant. There is broncophony on the right side above the clavicle and down to second rib. The whispered voice sounds are especially well reproduced and have an amphoric quality. Breath sounds above and below clavicle are amphoric. After coughing the rales have a resonating character. When in the upright position on looking over shoulders, the upper part of right lung seems to

move less than the left. Posteriorly the right lung is dull from apex to base, fremitus is feeble. Voice sounds have a slight ægophonic character. Breath sounds are feeble, bronchial in character, with numerous high-pitched crackling rales. Breath sounds beneath left clavicle are somewhat roughened. The pupils are equal, they react to light and to accommodation. The tongue is red and moist; the pulses are equal, regular, rapid, of high tension; arteries thickened. The heart sounds regular, rapid, strong, no murmurs. The temperature has ranged between 97° and 99° since admission. The sputum contained tubercle bacilli.

DISCUSSION.

Dr James Tyson: One statement of Dr. Allyn surprised me: that pectoriloquy, by which I mean direct detection of articulate speech, is an uncommon symptom in the presence of cavity. My own impression is that whispering pectoriloquy is quite common. Students often confuse—and sometimes physicians—bronchophony with pectoriloquy. Such confusion would make pectoriloquy more frequent. In my experience whispering pectoriloquy is not so rare in cavities.

Dr. S. S. Cohen: There is only one point to which I wish to refer beyond expressing my appreciation of the scholarly paper we have heard, and that is that the description of the so-called Wintrich's sign of cavity is rather misleading in regard to change of pitch. Of course, the comparison must be made under like conditions of respiration and strength of percussion. In other words, when there is a cavity communicating with a bronchus and so with the trachea and outer air the larger column of air set in vibration when the mouth is opened ought to give a lower note than that produced when the mouth is closed; and to my ear such is the case. The tympanic quality is of course more distinct, as usually stated. I have tested this again and again. At first I thought that my recollection of the classical description of Wintrich's sign was incorrect and refreshed my memory by going to the books. Like Dr. Tyson I find whispering pectoriloquy not at all uncommon.

Dr. R. N. Willson: I should like to call attention to the usefulness of a tuning-fork in locating and diagnosing a pulmonary cavity. It is a method upon which considerable has been written, but which has been used little. If a tuning-fork is set in vibration and its "handle-end" is carried from skin surface above the normal lung to that over the consolidation, and from the consolidation over the excavated portion distinct differences will be observed. This note obtained over a cavity will become suddenly very clear and musical. It is one of the very few reliable methods.

I have been interested in the great skepticism manifested by certain of

the Phipps Institute workers as to the possibility of diagnosing a cavity. They believe that more often a cavity is diagnosed incorrectly than correctly, and in watching the autopsies since my attention was called to their attitude I have come to the conclusion that they are in a measure correct. I think, however, the tuning-fork is a distinct help in determining the size and location of the cavity, and a procedure which is bound to render the diagnosis more feasible and accurate.

Dr. DAVID RIESMAN: Speaking of bell-tympany, Dr. Allyn remarked that it is not heard when the cavity is not very large, but only when the whole lung is involved. I have knowledge of a case in which the cavity was confined to the upper lobe, but in which there was, nevertheless, a beautiful coin-sound.

DR. ALLYN closes: Whether or not we get pectoriloquy is sometimes a question of our own ears. The voice sounds, especially the whispered voice sounds, are much altered, and one hears the sounds with greater clearness and nearness to the ear than over normal or consolidated lung. But in many instances I have been unable to convince myself that I could distinguish, aside from the sound that came through the air about the patient, *the exact words* which were spoken. We ask the patient to count one, two, three, and listen, and we are struck with the altered quality of sound and with how plainly the whispers come to us. I am not so sure, however, that in most instances we could tell what was said if we did not know beforehand the precise words which the patient was to utter.

DR. COHEN: I should like to ask Dr. Allyn some time when he has his class at Blockley, which affords abundant opportunity, to try the very experiment mentioned. The student not being allowed to know what the patient is asked to say, and using a binaural stethoscope to shut out the sound coming from the patient's mouth, the whispered voice will be so distinctly transmitted that he can hear and repeat the syllables.

APPENDIX.

ABSTRACT OF THE REPORT OF THE COMMITTEE ON MÜTTER MUSEUM, 1904.

The Committee of the Mütter Museum submitted their report for the fiscal year ending November 30th.

The arrangement of the museum has been carried on actively. During the current winter the study of the specimens has been made by more than one thousand students under the direction of Professor Frazier, of the University of Pennsylvania; Professor Rodman, of the Medico-Chirurgical College; Professor H. Augustus Wilson, of the Jefferson Medical College.

In the early part of the year the Pathological Society held an exhibition in the Hall and the adjoining rooms of the College. On Tuesday, December 1, 1903, a lecture was given by Dr. C. N. B. Carmac, of Cornell University College of Medicine, New York City, entitled "Aneurysm and Arteriosclerosis." The lecture for 1904 was given December 2, by Dr. George H. Monks, Assistant Visiting Surgeon, Boston City Hospital, and Lecturer on Surgery, Harvard Medical School, the Subject being "Studies in the Surgical Anatomy of the Small Intestines and Mesentery." Both lectures were attended to the full capacity of the room.

SUMMARY OF THE REPORT OF THE LIBRARY COMMITTEE FOR 1904.

The number of volumes now in the Library is (1909 more than were reported last year)	71,526

In addition:

Unbound Reports and Transactions number	6,807
Unbound Theses and Dissertations number	18,492
Unbound Pamphlets number	66,066
The General Library contains	54,856
The Lewis Library contains	13,284
The S. D. Gross Library contains	3,169
The Obstetrical Society Library contains	217

Three hundred and thirty-six dollars have been received from the sale of duplicates, besides a number that have been disposed of by exchange, but there are still 2858 on hand.

The Library receives regularly, by purchase or exchange, 442 medical periodicals—79 American and 363 foreign—32 more than last year; and in addition 208 American and 92 foreign current periodicals have been received. In most cases the exchanges of medical journals published in Philadelphia are sent to the Library through the courtesy of their editors.

Eight hundred and fifty-two dissertations have been received.

There have been received during the year, from all sources, 3889 volumes, 8920 pamphlets, and 36,921 numbers of medical periodicals.

Of the 626 new publications added to the Library, 32 were written or edited by Fellows of the College.

The "Donors" to the Library during the year number 287, of whom each of the following named presented 25 volumes or more:

Estate of Dr. T. M. Drysdale	958	volumes
Dr. T. D. Myers	211	"
Philadelphia Polyclinic	189	"
Dr. Francis R. Packard	152	"

Dr. Hobart A. Hare	144	volumes.
Dr. John B. Roberts	105	"
Mrs. V. Dunglison	45	"
Dr. Charles K. Mills	45	"
Dr. Arthur Van Harlingen	42	"
Dr. T. S. K. Morton	40	"
Dr. Rebecca Fleisher	37	"
Dr. H. H. Kynett	34	"
Dr. Charles Shaffner	32	"
Dr. George W. Norris	30	"
Pennsylvania Hospital	28	"

The Publishing House of

W. B. Saunders & Company has presented	30	"
F. A. Davis Company " "	14	"
P. Blakiston's Son & Co. " "	13	"
J. B. Lippincott Company " "	12	"
Lea Brothers & Company " "	7	"

The following gentlemen have presented a large number of pamphlets and journals: Drs. Allis, Atkinson, Davis, Dulles, Drysdale, Dunglison, Edsall, Fisher, Flick, Frazier, Gibb, Gould, Griffith, Hare, Harlan, Henry, Jopson, Keen, Kelly, Kynett, Martin, Mays, S. W. and J. K. Mitchell, Morris, Neilson, Noble, Oliver, Packard, Rhein, Riesman, Sailer, Sajous, de Schweinitz, Scott, Swan, C. F., J. G., and J. J. Taylor, Van Harlingen, Wharton, White, Willson, Witherstine, and H. C. Wood, Jr; also the Pathological Society, the Philadelphia Polyclinic, and J. B. Lippincott Company.

The following amounts have been expended for the purchase of books by the various Funds:—

Henrietta Rush Fales Baker Fund, 52 volumes	$168 42
Luther S. Bent Fund, 14 volumes	25 04
William T. Carter Fund, 36 volumes	139 26
John D. Griscom Fund, 116 volumes	250 90
William F. Jenks Fund, 79 volumes	316 11
Oliver A. Judson Fund, 12 volumes	61 44
Wm. V. and John M. Keating Fund, 18 volumes	47 40
W. W. Keen Fund, 66 volumes	137 95
Library Endowment Fund, 168 volumes	665 74
Weir Mitchell Fund, 64 volumes	337 23
Philadelphia Medical Society Fund, 7 volumes	40 25
Lewis Rodman Fund, 52 volumes	187 94
J. F. Weightman Fund, 22 volumes	66 03
Caspar Wistar Fund, 68 volumes	181 10

Special Accounts:

Fund for completing files of journals, 120 volumes		$269 75
Journal Association, 20 volumes . .	(about)	113 00
New Book Fund, 41 volumes		91 38
J. Ewing Mears Account, 29 volumes . . .		105 55
S. D. Gross Library, 47 volumes		172 35

One thousand two hundred and fifty-five volumes have been bound—217 more than last year.

The number of visitors to the Library was 6467—an increase of 217. Number of books taken out, 4885—1372 more than in the preceding year; 18,624 books were supplied by the Librarian for consultations in the Library—an increase of 1528 over last year, and a large number were taken directly from the shelves by Fellows of the College.

The cataloguing work is progressing satisfactorily. All new books and more important pamphlets have been catalogued and the cataloguing of the Hough collection and of the Norris Library has been completed. 21,739 cards have been revised—2175 more than during the last year. All the money collected for the purpose of revision has been expended and additional subscriptions are in order.

As usual the Library has been thoroughly cleaned during the summer and all the books have been examined and compared with the list. Heretofore we have been able to report that not a book has been missing, but this year three cannot be found. As they are not books that would be likely to be stolen, it is hoped that they have been mislaid in the crowding and shifting and may turn up safely at some future time.

Great credit is due to the employés of the Library for maintaining so well the order and accessibility of the books and the usefulness of the Library in spite of its overcrowded condition. Their work is being done under most unfavorable and discouraging conditions, but is being done well.

George C. Harlan, M.D.,
Chairman.

LIST OF PAPERS: SECTION ON OPHTHALMOLOGY.

December, 1903.

A Case Illustrating the Inadequacies of the Present Methods for the Recognition of Distant Color Signals, by Dr. Charles A. Oliver.

Simple Glaucoma in the Young, by Drs. C. A. Veasey and Edward A. Shumway.

A Case of Obstruction in the Central Retinal Artery, by Dr. Wm. T. Shoemaker.

A Case of Plugging of the Lower Temporal Artery, by Dr. James Thorington.

Temporal Hemianopsia, with Skiagraphic Examination, Indicating the Position of the Supposed Lesion, by Dr. G. E. de Schweinitz.

A Case of Hyaline Masses in Corneal Scar Tissues, by Drs. G. E. de Schweinitz and Edward A. Shumway.

A Case of Chronic Chorioretinitis and Extensive Pigmentary Degeneration of the Retina, by Drs. G. E. de Schweinitz and Edward A. Shumway.

Hallucinations of Vision, by Dr. H. F. Hansell.

January, 1904.

Coloboma of the Optic Nerve, by Dr. John T. Carpenter.

Palsy of the Extraocular Muscles in Exophthalmic Goitre, by Dr. Wm. Campbell Posey.

Concerning Certain Neuroretinal Lesions in Syphilis, by Drs. G. E. de Schweinitz and Howard Mellor (by invitation).

Some Notes on Keratitis Bullosa, by Dr. H. F. Hansell.

Prognostic Significance of Retinal Affections in Disease of the Kidney, by Dr. John T. Carpenter.

February, 1904.

A Case of Postneuritic Optic Atrophy Associated with Facial Paralysis, by Dr. Edward A. Shumway.

A Case of Abnormal Development of the Iris, by Dr. William Zentmayer.

A Case of Bilateral Enlargement of the Lacrymal Glands, by Dr. Wm. T. Shoemaker.

A Case of Fatal Septic Meningitis following Facial Erysipelas, by Dr. John T. Carpenter.

A Case of Dermoid of the Orbit, by Drs. G. E. de Schweinitz and Edward A. Shumway.

Associated Movement of Eyelid and Jaw, by Dr. G. C. Harlan.

March, 1904.

A Case of Sarcoma of the Left Cerebellum, by Dr. Charles A. Oliver.

A Case of Spring Conjunctivitis Resembling Malignant Growth of the Corneal Limbus, by Dr. C. A. Veasey.

A Case of Graves' Disease Exhibiting Unilateral Exophthalmos, by Dr. Wm. Campbell Posey and Dr. Walton C. Swindells (by invitation).

External Appearances of Chronic Uveitis from Interstitial Keratitis, by Dr. Charles A. Oliver.

One of the Dangers in the Use of Powerful Magnets, by Dr. S. D. Risley.

A Case of Inflammatory Glaucoma, by Dr. S. D. Risley.

A Case of Bullet Removed from the Orbit after Localization by the X-rays, by Dr. H. F. Hansell.

April, 1904.

Histological Examination of the Eyes in a Case of Amaurotic Family Idiocy, by Dr. Edward A. Shumway and Dr. Mary Buchanan (by invitation).

Traumatic Perforation of the Macula and Rupture of the Choroid, by Dr. G. E. de Schweinitz.

A Case of Atypical Pigmentary Degeneration of the Retina, by Dr. G. E. de Schweinitz.

A Case of Congenital Exophthalmos, Produced by Orbital Hemorrhage, followed by Metastatic Choroiditis, by Dr. C. A. Veasey.

A Case of Gunshot Wound of the Orbit, by Dr. Wm. Campbell Posey.

A Case of Filamentous Keratitis, by Dr. William Zentmayer.

A Case of Extensive Distribution of Cholesterin, by Dr. William Zentmayer.

October, 1904.

A Case of Pseudoglioma, by Dr. S. D. Risley.

A Case of Parinaud's Conjunctivitis, by Dr. Wm. Campbell Posey.

A Case of Parinaud's Conjunctivitis, by Dr. A. G. Thomson.

Conglomerate Tubercle of the Choroid, by Drs. G. E. de Schweinitz and Edward A. Shumway.

A Note on Some Forms of Muscular Inco-ordination, by Dr. H. F. Hansell.

November, 1904.

Determination of the Various Forms of Anomalies of the Ocular Muscles, by Dr. Alexander Duane, of New York (by invitation).

WM. M. SWEET,
Clerk of Section.

LIST OF PAPERS: SECTION ON OTOLOGY AND LARYNGOLOGY.

December, 1903.

Dr. Cornelius G. Coakley, of New York, read a paper entitled "Personal Experiences in the Diagnosis and Treatment of Diseases of the Accessory Sinuses of the Nose."

Dr. Frank Woodbury reported "A Case of Peculiar Affection of the Throat, Illustrating the Value of Antisyphilitic Treatment in a Condition which had been Diagnosed Epithelioma."

January, 1904.

Dr. Joseph S. Gibb presented "A Case of Suspected Lupus of the Soft Palate."

Dr. George M. Marshall read a paper entitled "Nose Deformity due to Fractures of Long Standing." Presentation of three cases corrected by operation.

Dr. Frank Woodbury reported "Some Remarks on Nasal Hemorrhage," and exhibited a specimen of myxoma removed from the septum narium.

February, 1904.

Dr. W. G. B. Harland addressed the Section on "A Case of Hemorrhage following Tonsillotomy."

Dr. H. Holbrook Curtis, of New York, read a paper entitled "Recent Methods of Treatment of the Accessory Sinuses."

March, 1904.

Dr. D. Braden Kyle presented "A Case of Subglossal Neoplasm."

Dr. W. G. B. Harland reported "A Case of Hemorrhage following Tonsillotomy."

Dr. G. B. Wood read a paper on "The Production of Leukocytes by the Tonsils," and exhibited some microscopic slides to illustrate his paper.

April, 1904.

Dr. B. A. Randall presented some "Remarks on Heredity in Ear Diseases."

Dr. Joseph S. Gibb discussed "Sepsis and Asepsis in Intranasal Surgery."

Dr. D. Braden Kyle addressed the Section on "Suppurative Mastoid Disease without Apparent Middle Ear Suppuration."

May, 1904.

Dr. P. S. Donnellan reported "Results of Operation in Three Cases of Acute Suppurative Disease of the Mastoid, Complicating Influenza." Presented patients.

Drs. Harland and Weisenburg presented "A Case of Hemiplegia of the Same Side following Mastoid Operation."

Dr. A. W. Watson reported "A Case of Antral Disease."

October, 1904.

Dr. E. L. Vansant presented some cases for examination.

Dr. G. Hudson Makuen read a paper entitled "The Causes of Defects of Speech," with exhibition of patients.

November, 1904.

Dr. Wendell C. Phillips, of New York, addressed the Section on "Inflammatory Manifestations in the Upper Air-passages due to Malarial Plasmodium."

Dr. W. G. B. Harland read a paper entitled "Verruca Obstructing the Nostril in a Colored Child."

Dr. G. B. Wood presented "A Case of Antral Neurosis."

Dr. J. K. Knorr (by invitation) showed "A Case of Papilloma of the Larynx."

Dr. Walter Roberts reported "A Case of Malignant Adenoma of the Larynx."

Dr. A. W. Watson showed "A Case of Sarcoma of the Antrum Ten Years after Operation."

FRANCIS R. PACKARD,
Clerk of Section.

LIST OF PAPERS: SECTION ON GENERAL MEDICINE.

Asthma of Gastric Origin, by Dr. Joseph Sailer.

Appendicitis--the Case which Completed My Conversion, by Dr. James Tyson.

Two Years' Experience in Gastroptosis, by Drs. J. Dutton Steele and A. P. Francine.

The Pathological Anatomy of Cirrhosis of the Liver, by Dr. W. W. Ford, of Baltimore (by invitation).

The Clinical Chemistry of Cirrhosis of the Liver, by Dr. David L. Edsall.

The Clinical Manifestations of Cirrhosis of the Liver, by Dr. Charles G. Stockton, of Buffalo (by invitation).

Remarks on the Uncinate Group of Fits and on Extensive Hemorrhage Occurring in Epilepsy, by Dr. William G. Spiller.

Persistent Seborrhœa following Herniotomy, by Drs. David Riesman and F. T. Stewart.

The Relation of Typhoid Fever to Tuberculosis, by Dr. J. M. Anders.

Some Remarks on a Severe Case of Appendicitis Ending in Recovery without Operation, by Dr. Herman B. Allyn.

The Diagnosis of Incipient Phthisis, with Special Reference to the Physical Signs and the Frequency of Bacilli in the Sputum, by Dr. Robert N. Willson.

A Preliminary Report upon Some Experimental Nutritive Enemata, by Dr. Chas. B. Wordon.

A Few Remarks about the Significance of Right-sided Pleurisy or Pneumonia in Subphrenic Abscess and Abscess of the Liver, with Report of Cases, by Dr. J. A. Scott.

A Note on Chronic Parotitis, by Dr. J. H. Musser.

The Pharmacological Action of Mercury; Its Influence on Autolysis, by Drs. D. L. Edsall and Caspar W. Miller (by invitation).

Some Clinical Observations on Cirrhosis of the Liver, by Dr. Alfred Stengel.

Hemorrhagic Diathesis in Bright's Disease; a Report of Two Cases, by Dr. David Riesman.

Cases Reported.

Two Cases of Uncinaria (Old World Parasite), by Dr. J. Norman Henry.

A Case of Acute Hemorrhagic Leukæmia, by Drs. Jas. B. Wister (by invitation) and Norman B. Gwyn.

Some Cases Showing Lesions of the Cardiac End of the Stomach, by Dr. Joseph Sailer.

A Preliminary Report of a Case of Splenomyelogenous Leukæmia Showing Marked Improvement under the Use of the X-ray, by Dr. Charles H. Weber.

A Case of Chronic Poisoning by Bisulphide of Carbon, by Drs. Henry D. Jump and John M. Cruiee.

A Case of Miliary Tuberculosis which Gave a Positive Widal Reaction, by Dr. Joseph Sailer.

A Case of Uncinariasis, by Drs. M. H. Fussell and C. A. Fife.

A Case of Cancer of the Œsophagus Simulating Thoracic Aneurysm, by Dr. J. A. Scott.

Patients Shown.

A Man with Signs of Mediastinal Tumor, the Nature of which Had Not been Definitely Determined, by Dr. Joseph Sailer.

A Patient with Chronic Cyanotic Polycythæmia, by Dr. Wm. Egbert Robertson.

A Case of Cervical Hernia of the Lung of Spontaneous Origin, by Dr. Clifford B. Farr.

A Patient with Pulsating Empyema, by Dr. Wm. B. Stanton.

A Case of Phthisis of the Base of the Lung, by Dr. A. P. Francine.

A Patient Illustrating the Curative Effects of the X-ray in Hodgkin's Disease, by Dr. J. A. Scott.

Pathological Specimens Presented.

A Large Uric Acid Calculus, which had been passed per urethra, weighing seventeen grains, by Dr. Henry Fisher.

A Specimen of the Parasite of Uncinariasis, by Drs. M. H. Fussell and C. A. Fife.

Post-mortem Specimens from a Case of Cancer of the Œsophagus Simulating Thoracic Aneurysm, by Dr. J. A. Scott.

S. McC. Hamill,
Clerk of Section.

LIST OF PAPERS: SECTION ON GYNECOLOGY.

December, 1903.

Immediate Repair of Lacerations following Labor, by Dr. Striker Coles.

Pelvic Cellulitis, by Dr. R. P. McReynolds.

Cystic Carcinoma of the Ovary, an Ovarian Cyst, and an Extrauterine Pregnancy (case reports), by Dr. G. E. Shoemaker.

February, 1904.

The Defences of the Body and Syncytial Invasion, by Dr. Joseph McFarland (by invitation).

April, 1904.

Tuberculosis of the Vermiform Appendix, Retroperitoneal Lymph Glands, and Fallopian Tubes, by Dr. E. A. Schumann (by invitation).

A Case of Adenomyoma of the Undeveloped Horn in a Uterus Bicornis, by Dr. B. M. Anspach (by invitation).

October, 1904.

A Case of Complicated Inversion of the Uterus, by Dr. B. M. Anspach (by invitation).

A Clinical Study of Seventy-five Cases of Ectopic Pregnancy, by Dr. B. C. Hirst.

Recent Theories Concerning the Origin of Glandular Ovarian Cysts, by Dr. John G. Clark.

A Case of Carcinoma of the Vulva, by Dr. John H. Girvin.

Technique of the Operation of Ventrosuspension, by Dr. H. D. Beyea.

WILLIAM R. NICKOLSON,
Clerk of Section.

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